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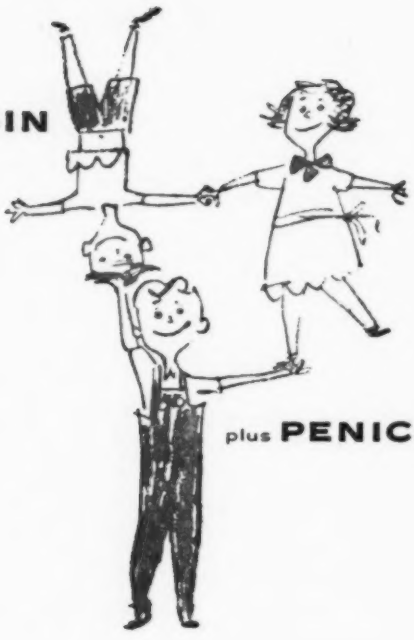

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**Crump, E. P.: Postgraduate Med., 13:552, 1953*

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The Journal of Pediatrics

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POLIOMYELITIS APHORISMS

PHILIP M. STIMSON, M.D.
NEW YORK, N. Y.

POLIOMYELITIS is a variable disease, varying in severity from case to case, from epidemic area to epidemic area, from year to year. What happens in one patient does not happen in the next. But many symptoms, signs, and responses to treatment happen in a high enough percentage of cases to be significant, and doctors who see and treat large numbers of patients with poliomyelitis come to many common conclusions. In this paper an attempt is made to express in dogmatic form and without appreciable discussion a number of observations and conclusions on which most poliomyelitis authorities agree.

Poliomyelitis should be considered as contagious as measles, and as with that disease, transmission is thought to be mainly by contact, such as by familial spread.

The period of maximum communicability corresponds closely with the period when the virus is demonstrable in the pharynx, that is, the brief period of a few days before and after the onset of symptoms.

The virus is present in the blood stream the last few days of the incubation period, even in patients who never develop recognizable polio. Viremia does not persist after the first day of fever.

The virus is present in the stools of patients and of contacts for an indefinite period, but with proper sewage disposal this is not a considerable hazard. Outdoor privies and flies can add to the spread of polio.

In countries with primitive sanitary conditions, fecal infection is more common and a large proportion of the population is repeatedly exposed. Babies receive congenital antibodies from their mothers, and while thus protected and afterward, are repeatedly exposed, sometimes contracting the disease, but in any case developing immunity with or without obvious infection. Thus polio becomes rare after infancy in such countries.

Twenty-four hours after the temperature has returned to normal, the patient may be considered noncontagious if living under proper sanitary conditions of sewage disposal.

It is probably common to be a healthy virus carrier, at least for a short time. A healthy carrier may have his equilibrium of resistance upset by a number of factors which can be divided into two groups. In the first group are factors that facilitate introduction of the virus into the body, such as the trauma of nose and throat operations and of tooth extractions. In the second group are factors that increase the severity of an already established infection such as excessive

From the Department of Pediatrics, New York Hospital-Cornell University Medical College. Read before the New Orleans Graduate Medical Assembly, March 9, 1954.

fatigue or chilling, pre-existing infections such as of the respiratory tract, pregnancy, cortisone, local tissue insults such as certain inoculations, and, finally, perhaps high atmospheric temperatures, adulthood, and mental stress. Most of all, resistance probably depends upon the presence of neutralizing antibodies.

The diagnosis of acute polio without paralysis is a presumptive one, based on evidence of involvement of the central nervous system shown by headache, fever, vomiting, some stiffness of the neck and back, irregular abdominal reflexes that have a tendency to disappear, occasional muscle tremors and twitchings, and usually, but not always, an abnormal spinal fluid. There is no relationship between the number of cells in the spinal fluid and the extent or severity of the muscle weaknesses.

Patients showing enough of the above manifestations to make a diagnosis of acute polio reasonably certain will often during the second week show some slight muscle weakness if careful search is made. The weakness may not last more than a few days, particularly if the patient is given complete bed rest.

Tremors of the hands when holding a glass of water at arm's length may foretell oncoming weakness in an arm. Twitchings of scattered muscle groups are not uncommon in early poliomyelitis.

In a number of diseases, such as polio, the various encephalitides, and acute lymphocytic choriomeningitis, the spinal fluid shows the same findings, consisting of a moderate increase of white blood cells, particularly mononuclear cells, and a slight increase of protein with a normal sugar content. There is no relationship between the number of cells in the spinal fluid and the extent or severity of the muscle weaknesses.

In certain epidemics of polio, a normal spinal fluid can be obtained in 10 to 15 per cent of the cases; hence, the main value of the examination of the spinal fluid in a suspected case of polio is to rule out the presence of septic meningitis. An abnormal fluid indicates only the presence of some irritation of the central nervous system, one which might be polio or something else. A normal spinal fluid does not rule out the diagnosis of acute polio.

TABLE I. POLIOMYELITIS. NUMBER OF CASES IN A FAIRLY SERIOUS EPIDEMIC (100+ CASES PER 100,000 POPULATION) FOR EACH 1,000 PEOPLE

1	case of acute polio with permanent weakness
1	case of acute polio with temporary weakness
3 to 10	recognizable cases of acute polio without weakness
10 to 25	cases of "characteristic minor illness"
100 to 500+	cases of healthy carriers

Eventually possibly 90 per cent of adults will have neutralizing antibodies.

A patient showing mild manifestations suggestive of polio, but in no way suggestive of meningitis, may well be spared the ordeal of a lumbar puncture and spinal fluid examination.

If a person with a suspected case can kiss his knees, it is probably not poliomyelitis.

A symptom complex otherwise suggestive of early poliomyelitis is probably not that disease if any one of the following manifestations is present: (1) "point tenderness," easily demonstrated by pressure with the eraser end of a pencil; (2) loss of sensation; (3) a septic type of temper-

ature, especially if over a week in duration; (4) a white blood count higher than 13,000; (5) local swelling, as of a joint or muscle; (6) a frozen joint in which no motion can be obtained; (7) severe pain at rest. Headache, backache, leg or arm aches, sore throat, all are common, and motion causes pain in the adjacent muscles, but when lying still the poliomyelitis patient is not especially uncomfortable.

Abdominal pain may appear early in polio, perhaps due to congestion of mesenteric lymph nodes. Operations for a suspected appendicitis have been done under these circumstances.

Polio patients under one year of age, even though showing muscle weakness, have very little if any muscle tightness.

The commonest clinical condition to be mistaken for acute polio is influenza with headache, fever, vomiting, and general muscular pains.

Hysteria may be confusing in the diagnosis, but deep muscle reflexes are still present and the hysterical paralysis is likely to involve all the muscles normally engaged in a given function and not just scattered muscles here and there.

Generalized paralyses that sometimes occur late after a severe case of diphtheria may be differentiated from those of polio in that they are not accompanied by spasm or muscle pains and are usually symmetrical weaknesses rather than a total paralysis. The muscles of accommodation of the eyes are practically invariably involved in a diphtheritic paralysis, but almost never involved in polio.

The causes of breathing difficulty in polio are complex and should be thoroughly studied in each case and clearly differentiated. They fall into three groups. First are those involving the mechanism of breathing. The actual muscles used may be tight, or weak, or both, and the intercostals and the diaphragm may be separately or together involved. The respiratory center in the bulb may be involved so that the act of respiration is inadequately stimulated.

The second group of manifestations that cause breathing difficulty have to do with interference with oxygen and carbon dioxide exchange. This interference may be in the pharynx where a paralyzed tongue may fall back into the pharynx and cause some mechanical obstruction, or where, because of an inability to swallow, mucus and saliva may accumulate in the pharynx and cause a fluid obstruction. The larynx may be the source of trouble, either as the result of spasm as in choking, or a result of paralysis either of the abductor or adductor muscles of the vocal chords. In the trachea or bronchi there may be obstruction from the secretions of bronchitis or from inhaled saliva or vomitus which the patient cannot remove because of inadequate coughing. Until secretions are removed, artificial respiration is dangerous. In the lungs, obstruction to oxygen and carbon dioxide exchange may be caused by pneumonia or atelectasis or pulmonary edema. In the chest, circulatory difficulties due to myocarditis or right heart failure may be present. All this group of factors interferes with oxygen and carbon dioxide exchange.

The third group of factors causing breathing difficulty are contributory. The first of these is panic and hysteria. The second is sedation, unwisely given to an exhausted patient.

The third is anemia whereby there is an inadequate supply of hemoglobin to carry oxygen to the tissues. Fourth is air swallowing, particularly in a child who gulps air into his stomach while trying to draw the air into his lungs. A fifth factor is fatigue.

It is usual to have a number of these causes or factors active in any given case of breathing difficulty. The treatment will vary greatly with the causes found.

Proper treatment of patients with breathing difficulty includes a great deal of clinical knowledge, fine judgment, skill, and a minimum of four mathematical determinations. A doctor's appreciation of the usages of these four determinations is essential for the patient's best welfare. First is the blood pressure; the second, vital capacity; the third, tidal volume; and the fourth, carbon dioxide elimination.

Elevation of blood pressure is due to a number of causes, probably mostly originating in the central nervous system. Irritation of the vasomotor center in the bulb can cause increased blood pressure as also can anoxia. Most respirator cases have at least some elevation of blood pressure. Treatment to reduce the elevation, unless extreme, is usually not necessary, but falling blood pressure may mean approaching failure of the vasomotor center. For this, Neo-Synephrine given intravenously is probably the best remedy, but the prognosis of a rapidly dropping blood pressure is often bad.

If a patient's observed vital capacity is 50 per cent or less of his predicted vital capacity, he probably will do well with a little artificial respiration for a while to give him rest and sleep. If below 35 per cent, he probably needs continuous artificial respiration.

An adult whose vital capacity is down to 1,500 c.c., and dropping, probably cannot sleep without artificial respiration. At 1,200 c.c. he needs help.

After the acute stage is past, when the patient's temperature has returned to normal, and he has become less tense and worried, a man who at 1,200 c.c. needed artificial respiration, may be able to breathe without the respirator even though his vital capacity is only 600 or 700 c.c. A patient who is being weaned from a respirator, and while out of the respirator has a decrease in his vital capacity, as from 800 to 700 c.c., has become too tired. On the other hand, if his vital capacity has remained the same or has increased, he can perhaps stay out a little longer. With beginning exercise the muscle activity increases again the need for oxygen.

Tidal volume, or the air exchange in one breath, is of value in the comparison of forms of artificial respiration, or of the adequacy of any one type. In each breath the amount of useful air is that which reaches the lungs, the air in the mouth, trachea and bronchi being useless for ventilation. This dead space is from 100 to 150 c.c. in adults, so that if the tidal volume is 250 c.c., only about 125 c.c. is useful. To increase the amount of useful air reaching the lung in a minute, it is better to increase the size of each breath than to increase the rate of breathing. The tank respirator will do the former; the unaided patient with polio is most likely to do the latter.

Measuring lung alveolar carbon dioxide tension is the best method of

determining whether carbon dioxide is being properly eliminated or not. The equipment for doing this is usually not available. The next best method is by arterial measurement. This requires aspirating a femoral artery, which is not always possible. Venous plasma carbon dioxide combining power measurements are better than none. When such carbon dioxide is retained above a normal of 62 vol. per cent, respiratory acidosis is present with usual symptoms of headache, dizziness, even delirium and coma. The cure lies in increasing the ventilation by increasing the respiratory excursion, thereby "blowing off" the excess carbon dioxide by temporary overventilation.

There still is no cure for the polio infection except time.

The most important measure of treatment in the acute stage is natural sleep. Artificial sleep, induced by sedation, is dangerous in acute cases, lest breathing difficulty develop. A polio patient with fever who is asleep should be awakened for nothing at all, neither rounds nor meals nor treatments, and particularly not to have his temperature taken or his face washed at 5 A.M. The acutely ill patient should not be fatigued by undue treatments, examinations, or visitors. He should not be hurt. He should be calmed, sympathized with, helped to relax in mind and body. A mother should be encouraged to read aloud to the patient; it helps to keep the child quiet, and the mother busy. Prone packs are very comforting.

For apparently mild cases there is little danger in leaving the patient home for another eighteen to twenty-four hours to watch developments, and there are many good reasons for doing so. A fatiguing trip to the hospital will do him no good and may well aggravate the oncoming polio. Transportation to a hospital, when indicated, should be done with a minimum of physical and nervous fatigue to the patient.

There are certain clear indications for the removal of a patient to a suitable hospital. A suitable hospital is one equipped with the "know-how" and with teamwork, even more importantly than with mechanical equipment which can be rushed in. The removal indications include: increasing fever, marked prostration or an appearance of being really sick, any suggestion of breathing difficulty, difficulty in swallowing, a difficulty in emptying the bladder, and weakness of a deltoid muscle.

If one deltoid muscle is weak, there is likely to be some weakness of the diaphragm. If both deltoids are weak, there almost certainly will be extensive diaphragmatic weakness and breathing difficulty.

In the treatment of polio the indications for antibiotics are first an appreciable elevation in the white blood cell count, which suggests the presence of a secondary bacterial infection; second, the necessity to catheterize a patient, which makes a bladder infection likely, and third, the presence of breathing difficulty and with it a liability to secondary pulmonary infections.

For the treatment of the cramps and muscle twitchings that occur during acute polio, quinine is commonly effective.

Of patients dying in the acute stage, over half the hearts at autopsy will reveal more or less myocarditis. The polio virus has been found in heart

muscle. Acutely ill polio patients must also be considered as at least potential cardiac cases.

During the febrile period and for some days or weeks afterward, most polio patients cannot empty their bowels, and catharties only serve to cause abdominal cramps. Enemas should be used at least every other day, oftener if necessary, to avoid fecal impaction. Enemas are more effective if a snug abdominal binder is first applied.

In polio, artificial respiration can be given by a number of ways. First by the use of a tank respirator, the most effective means of ventilation and the only one that should be used in the acute stage of the breathing difficulty; second, by means of a rocking bed which makes the diaphragm move up and down like a bellows. The third method is by means of a so-called Cuirass respirator, which is applied to the trunk and has its action also principally on the diaphragm. A Cuirass is useful for transportation for short distances and for artificial respiration during such operative procedures as tracheotomy. A fourth method is by positive pressure over the face administered either by a face mask or by the hood on a tank respirator. This is of value when used for a patient whose tank respirator must be opened for some procedure, and even also to permit Hubbard tank treatments.

In emergencies it is possible to give artificial respiration by means of blowing periodically into a face mask which is held over a patient's face.

It is better than the collar of a tank respirator leak a little around the patient's neck rather than be tight and obstruct venous drainage from the head.

A stethoscope held over the mouth of a patient in a respirator can give information as to the presence of moisture in the bronchial tree, the presence of laryngeal obstruction, or perhaps even of adequate ventilation.

Within four to six weeks in a tank respirator the chest is likely to become frozen. Weaning is quite a different proposition when tried late, instead of early.

Occasional forced deep breaths are indicated for all patients with tight chests, as are also daily short periods of hyperextension and of flexion of the spine, and any other procedure that will tend to loosen up the costovertebral articulations.

The rocking bed causes artificial respiration by the effect on the diaphragm which rises when the head of the bed falls, and sinks when the head of the bed is elevated. This is useful for mild cases of breathing difficulty and, most particularly, for the help of patients who are being weaned from a tank respirator. The tidal volume obtained by a rocking bed is not as large as that obtained by a tank respirator. Patients on a rocking bed should have no pillow behind the head so that it may be in a line with the body and there should be only a slight angle at the waist so as to leave the chest and abdomen in the optimum position for the diaphragmatic movement. A small pad between the shoulders helps to expand the chest. The extremities should all be put into positions of abduction for several periods daily to mobilize the joints. Patients can also be placed prone on a rocking bed, the artificial respiration in this position being 90 per cent as efficient as for the supine position.

A patient on a rocking bed who is being weaned from a tank respirator may feel and look hot with the sudden improvement of circulation, particularly in the lower extremities. Venous return is aided, kidney function is improved, and the tendency to renal stones is apparently lessened, as is the case when a tilt table is used.

A patient should not be kept on a rocking bed long enough to develop a feeling of fatigue, as shown, for instance, by a more rapid pulse and restlessness, or a decrease of vital capacity.

This type of artificial respiration is hampered by a distended abdomen or by a flabby abdominal wall. It should not be used for patients with bulbar paralysis, particularly those unable to swallow. It should not be used for patients with marked breathing difficulty unless a tank respirator is not available.

The initial use of a rocking bed should not be after a meal. However, patients soon learn to eat meals on the bed and to sleep while it is rocking.

Runabout children should not be allowed near a rocking bed in motion lest they be injured.

A rocking bed needs to be balanced. Usually the start down of the head-end requires the maximum current, but can be helped by adding weight to that end.

In bulbar poliomyelitis, patients with no other breathing difficulties than an inability to swallow or to cough are much more comfortable and able to breathe much better if given postural drainage and suction. For this they are placed on their sides with the foot of the bed moderately raised. The head should be placed on a small pillow that keeps it in a line with the spine. The upper arm and leg should be rested on large pillows. Frequent suction should be used or the patient should be told to leave his mouth open and let the mucus run out into a kidney basin or wad of gauze. Some can suction themselves, and, when possible, should be encouraged to do so.

Bulbar cases who, because they are unable to swallow, have taken no fluids and have lost by suction what saliva they have produced, require the replacement by vein both of fluids and of electrolytes, given not more rapidly than a half liter an hour.

Patients unable to swallow must be fed by vein at least until the fever has returned to normal. About the seventh or eighth day of the disease, a plastic feeding tube passed through the nose can sometimes be extended into the stomach without distress to the patient or inducing vomiting. The patient can then be fed by tube but at the time should be gatched up in bed to prevent the fluids rolling out of the stomach through a relaxed cardia into the esophagus and pharynx.

When a patient seems able to swallow his own saliva, very small amounts of water may be offered by teaspoon at half-hour intervals, increasing gradually. Milk is not well tolerated the first few days after swallowing returns. When the patient is again able to eat somewhat, he should not eat or drink alone, because of the danger of choking as a result of the food or fluid entering a relaxed larynx. If choking does occur the patient should be placed face down on his bed with the hips elevated, and then patted on the back.

In bulbar cases the temperature regulatory center may become involved and very high temperatures may be seen in terminal cases.

The indications for tracheotomy in polio depend in most instances on the skill and amount of the trained personnel available. It is possible by expert handling, including skillful suctioning, bronchoscopy, and nursing care, to avoid tracheotomy which would be lifesaving in the absence of such skill. Tracheotomy, however, is always necessary when there is abductor paralysis of the vocal cords, allowing them to come together to cause a mechanical barrier to breathing. Every case of bulbar polio, particularly involving the larynx, should be watched by a laryngologist to note the range of motion present in the vocal cords, although the condition of the voice may help to indicate the state of the vocal cords.

A second indication for a tracheotomy is in a patient with bulbospinal polio who cannot cough or swallow, and who has to be put in a respirator. It is very difficult to keep a trachea clear of mucus except through a tracheotomy tube when the patient is in a respirator.

Suction through a tracheotomy tube should not be assigned to unskilled hands without careful instruction. Rough suctioning can cause increase of mucus.

Some clinics do a tracheotomy on every patient with bulbar polio whether or not there is breathing difficulty. In the absence of any breathing difficulty it should be possible to keep the pharynx clear of mucus by means of postural drainage, suction, and reasonable nursing care, without having to subject the patient to tracheotomy. Similarly, a spinal case with breathing difficulty but with no difficulty in swallowing should not have to have a tracheotomy done.

When a tracheotomy tube is worn a long time, a polyp may grow inside the trachea near the site of the tracheotomy, and should be looked for before permanently removing the tube.

About a fourth of respirator cases develop gross atelectasis, either while in a respirator or afterward. The easiest way of detecting gross atelectasis in a patient in a respirator is by noting a change in the position of the apex of the heart, which will move markedly to the side of the collapse.

Atelectasis was formerly best treated by immediate bronchoscopy and removal by suction of a possible mucous plug. Today it is best treated by making the patient have a series of artificial coughs. For instance, Barach's exsufflator with a facial mask and positive pressure expands the chest markedly and then suddenly allows it to collapse. This same action can be accomplished to a lesser degree on a patient in a tank respirator by hitching an ordinary vacuum cleaner to a porthole of the tank respirator and, while constant suction is being exerted, breaking the suction periodically by suddenly opening the bedpan porthole for four or five seconds. Momentary suction applied by Barach's exsufflator during the recoil expiration can continue the excursion to negative intrathoracic pressure, even to surpassing the flow rate of a normal cough. Such exsufflation with negative pressure is efficient even in the removal of some foreign bodies, as well as in the removal of mucous plugs and secretions from the tracheobronchial tree.

An over-all course of treatment for a case of polio might be outlined as follows:

1. Rest, as much natural sleep as possible, avoidance of any fatigue for up to three weeks.

2. The prevention or removal of contributory factors. These factors are:

a. Tightness of muscles, and pain.

b. Inadequate ventilation and respiratory obstruction.

c. Secondary infections, especially respiratory.

d. Anemia or dehydration.

e. Abdominal difficulties, b o w e l, bladder, distention.

f. Panic, worry.

3. Achieve adequate vital capacity.

4. Physiotherapy to achieve normal range of motion at each joint and to prevent contractures.

5. Exercise to achieve muscle control and coordination.

6. Exercise for strength.

7. Train for best use of what is left (with mechanical aids) to attain nearest approximation of normal life.

8. Finally, throughout the treatment instill patience, the realization that everybody has some restrictions, the ambition to make the most of what one has, and thoughtfulness of others.

TABLE II. POLIOMYELITIS. END RESULTS OF EVERY 100 CASES WITH WEAKNESS IN THE ACUTE STAGE

50 to 60	will recover completely
20 to 30	will always have some weakness, but comparatively few restrictions
5 to 10	will be seriously restricted, and probably require some help in daily living
2 to 3	will need more or less artificial respiration indefinitely
4 to 10	will die in the first month

A paralytic polio patient who has efficient treatment in the first or second stage of the disease should have no appreciable deformities such as those that are due to contractures. Paralysis per se is often less crippling than are contractures which are almost always preventable. After fever has gone, exercises should be instituted to relieve tightness of the muscles until a normal range of motion can be obtained at all joints, regardless of whether or not there is weakness, but not if weakness is so severe as to make a return of function hopeless. Sometimes a persistent tightness as of the spine is necessary for stability.

A patient with obvious restrictions should be weaned from institutional care by gradual steps: first a short trip out, or a meal out, then a week-end home. After such a visit home, he is asked, "What did your family need to do for you? Some of those things we will now try to teach you to do for yourself."

He might well be asked also, "And what did you do for others this week end?"

Because a thoughtful and considerate doctor can do much to help the patient—and the patient's family—to conquer with courage the afflictions of the disease, to rise with nobility above the restrictions, and with zest to become again a useful citizen, poliomyelitis is a disease that offers a challenge to any doctor to be more than just a dispenser of pills or a wielder of a knife—to be a guide, counselor, and friend. The rewards in gratitude and appreciation are boundless.

DEATH DUE TO ACCIDENTAL POISONING IN YOUNG CHILDREN

KATHERINE BAIN, M.D.
WASHINGTON, D. C.

A RECENT report[1] in the British literature on substances responsible for accidental poisoning in young children prompted a review of the situation in the United States. Though the comparison of death rates between countries is not always profitable, since conditions may not be comparable, it seemed desirable to look at the United States situation to see where it was similar and where it differed. As a result of this analysis of the United States figures some facts have appeared which were unexpected and which, in addition to being interesting, point the way for emphasis in prevention.

During the decade 1940 to 1950 in England and Wales, 218 children 1 to 5 years of age died of accidental poisoning.* On the logical assumption that the United States rate is approximately the same as the British, Veeder,[2] commenting editorially on Swinscow's article, estimated the number of deaths in the United States for the decade at 900. It is surprising, therefore, to find that instead of the expected 900 deaths from this cause there were more than 3,600 (see Table I).

The British rate for the period was 0.87 per 100,000 population. The United States rate was 3.6 per 100,000, more than four times as high. In the early years of the decade the contrast was even greater. Since 1945 the British rate has increased somewhat, while the United States rate has shown a slight decline. An even greater discrepancy between the rates for the two countries exists in the group under 1 year of age, the British rate being 0.29 per 100,000, in contrast to a United States rate of 1.8. The higher United States rate for this particular cause not only holds throughout childhood but is found at all age levels.*

In an effort to search for reasons for this difference, data for a two-year period, 1949 and 1950, for the age group under 5 years were analyzed in detail (see Table II). Differences in composition of the population groups might possibly account for these differences in rates. Since the sex distribution is assumed to be the same in the two populations, male to female ratios of death rates should not differ greatly. Deaths from accidental poisoning, like other childhood accidents, occur more frequently in males than in females in both countries. The United States sex ratio is 3:2 and the British figures are similar. Under 1 year the higher rate

From the Children's Bureau, Department of Health, Education and Welfare.

This paper is based on data from special tabulations supplied by the National Office of Vital Statistics, Public Health Service.

Presented before the 64th annual meeting of the American Pediatric Society, Buck Hill Falls, Pa., May 3, 1954.

*For the purpose of this paper only accidental poisonings due to solids and liquids are considered. For the years 1940 to 1948, these are included in the International List No. 179, Fifth Revision. Beginning in 1949, the Sixth Revision was in effect and these deaths are recorded under E870-E888.

*Some differences exist between the two countries in the form of the death certificate and through 1948 in coding primary cause, but these are minor and could not account for the great difference in rates.

for males in England and Wales is not duplicated in the United States, but small numbers may account for this apparent discrepancy.

Racial differences in the two populations might possibly account for differences in rates. The British population is almost exclusively white, while the nonwhite population in the United States in the group under 5 years is about 12 per cent of the whole group. Nonwhite death rates from accidental poisoning are three times higher than white rates but are not high enough to account for the entire difference between the two countries. The United States white rate (2.3) is still appreciably higher than the British (1.1). The high nonwhite rate probably reflects the low economic status of this group, rather than a true racial difference.

TABLE I. NUMBER OF DEATHS AND DEATH RATES PER 100,000 FOR ACCIDENTAL POISONING, UNDER 1 YEAR AND 1 TO 5 YEARS: UNITED STATES, 1940 TO 1950

YEAR	UNDER 1 YEAR		1 TO 5 YEARS	
	NUMBER	RATE	NUMBER	RATE
1940	44	2.2	333	3.9
1941	29	1.3	292	3.4
1942	37	1.6	354	3.9
1943	58	2.2	393	4.2
1944	52	2.1	437	4.4
1945	41	1.7	397	3.8
1946	38	1.6	408	3.8
1947	59	1.7	336	3.1
1948	55	1.7	350	3.0
1949	64	2.0	359	2.9
1940-1949	477	1.8	3,659	3.6

Note: Deaths in 1940-1948 classified by the Fifth Revision of the International List of Causes of Death (List No. 179) and in 1949 by the Sixth Revision (List No. E870-E888).

TABLE II. DEATH RATES AND NUMBER OF DEATHS FROM ACCIDENTAL POISONING, BY RACE AND SEX, UNDER 5 YEARS, UNITED STATES, 1949-1950

AGE GROUPS	TOTAL	RACE		SEX	
		WHITE	NONWHITE	MALE	FEMALE
	Annual rate per 100,000				
Under 5 years	2.6	2.1	6.2	3.1	2.1
Under 1 year	1.8	1.4	4.5	1.8	1.7
1 to 5 years	2.8	2.3	6.7	3.4	2.2
	Number of Deaths (2 years)				
Under 5 years	834	591	243	499	335
Under 1 year	113	78	35	59	54
1 to 5 years	721	513	208	440	281

It is in the kinds of substances responsible for the deaths that one looks for clues to the reasons for the differences between the two countries.

The substances which children ingest are of so many kinds that grouping of related substances is necessary for comparison. Swinscow[1] employed a useful classification, as follows:

1. Medicaments
2. Disinfectants and antiseptics
3. Household poisons
4. Poisonous plants
5. Miscellaneous

This classification cannot be duplicated exactly for the United States material, but a somewhat similar grouping can be used:

I. Drugs
II. Petroleum products
III. Materials intended for external use
IV. Noxious foodstuffs
V. Unclassified

(See Table III for details of grouping)

There are obvious difficulties in such a classification. The Sixth Revision of the International Lists—the use of which began in 1949—is designed to identify the poison and not the circumstances under which it was obtained. Arsenic, for example, might be obtained in a medicine or an insecticide, but all arsenic deaths are put in group III. The source of strychnine

TABLE III

	SIXTH REVISION OF INTERNATIONAL LISTS	
	SUBSTANCE	NUMBER
I. Drugs	Morphine and other opium derivatives	870
	Barbituric acid and derivatives	871
	Aspirin and salicylates	872
	Bromides	873
	Other analgesic and soporific drugs	874
	Sulfonamides	875
	Strychnine	876
	Belladonna, hyoscine, atropine	877
	Other and unspecified drugs	878
	Part of Other and unspecified solid and liquid substances	888*
II. Petroleum products	Petroleum products	881
III. Materials for external use	Alcohol	880
	Industrial solvents	882
	Corrosive aromatics, acids, and caustic alkalis	883
	Mercury and its compounds	884
	Lead and its compounds	885
	Arsenic, antimony and their compounds	886
	Fluorides	887
	Part of Other and unspecified solid and liquid substances	888*
IV. Noxious foodstuffs	Noxious foodstuffs	879
V. Unclassified	Part of Other and unspecified solid and liquid substances	888*

*Additional data for the years 1949 and 1950 have been obtained from death certificates assigned to List No. 888, other and unspecified solid and liquid substances, so that these cases could be placed in proper groups. A few fell in group I, most in group III, and the remainder were designated unclassified, since the substance responsible was given in vague terms such as "pill," "poison," "tablet." The number of such discards is recorded in Table IV.

TABLE IV. DEATH RATES AND NUMBER OF DEATHS FROM ACCIDENTAL POISONING, BY CAUSE, UNDER 5 YEARS, UNITED STATES, 1949-1950

CAUSE	TOTAL	WHITE	NONWHITE
	Annual rate per 100,000		
Total—1949 and 1950	2.6	2.1	6.2
I. Drugs	0.9	0.9	0.6
II. Petroleum products	0.7	0.4	2.3
III. Material for external use	0.9	0.7	2.8
IV. Noxious foodstuffs	—	—	—
V. Unclassified	—	—	—
	Number of deaths (2 years)		
Total—1949 and 1950	834	591	243
I. Drugs	276	251	25
II. Petroleum products	210	121	89
III. Material for external use	300	191	109
IV. Noxious foodstuffs	8	3	5
V. Unclassified	40	25	15

might be medicine or a rodent poison, but all cases are assumed to be in group I. Without additional information on each case, which is not now recorded on death certificates, some cases may be assigned to the wrong group, but it is assumed such errors are few.

this is the most prominent group, having about two-thirds of the cases. In the United States it is of relatively less importance, accounting for about one-third of the cases (see Chart I). The most common drug killer is aspirin (and the salicylates). Deaths from this cause numbered 113 under 5 years

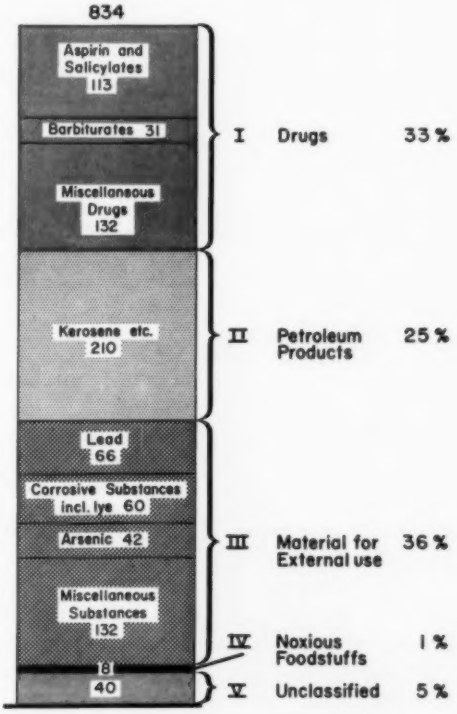

Chart I.—Substances responsible for death from accidental poisoning of children under 5 years: United States, 1949 and 1950.

The three main groups which contained nearly 95 per cent of the cases are analyzed in some detail (see Table IV).

DRUGS

This category corresponds in general to Swinscow's group called medicaments. In England and Wales

of age in the two-year period. Barbiturates are the next most lethal, accounting for 31 deaths. Both of these drugs are commonly found in the family medicine cabinet. Iron poisoning, common in England and Wales, is relatively rare here, only 2 cases being recorded in the two-year period.

Antihistamine deaths occur several times a year. One death due to Dramamine occurred in 1950.

PETROLEUM PRODUCTS

These are pulled out for a special group because of their number. More than 100 deaths in one year in the under-five-year group occurred in the United States from this cause, in contrast to 15 aged 1 to 5 years in a nineteen-year period in England and Wales. Kerosene is a common fuel oil in this country, especially for low-income families and for Negro families. The nonwhite rate for this cause is about six times the white rate. Poisoning from this cause is one of the major factors in increasing the United States rate over the British rate, and because of its volume makes drug poisoning appear relatively less important in this country than in England.

MATERIALS FOR EXTERNAL USE

Lead ranks highest in this category, and arsenic is still very important as a child killer. Caustic substances, such as lye, Lysol, and ammonia cause about 30 deaths annually. The rat poisons and fly sprays continue to be hazards to young life. The list of "other" items contains a wide variety of substances such as permanent wave solution, shampoo, lighter fluid, rubbing solution, bleaching agent, antifreeze, moth balls, and furniture polish, all common household substances. Some of the newer insecticides and pesticides, such as DDT and Parathion, are appearing in the list.

NOXIOUS FOODSTUFFS

Few deaths are reported in the United States as due to this cause. The 8 in 1949 and 1950 occurred in Mississippi, Georgia, Florida, and North Carolina, and were recorded as due to such substances as "wild berries," "toadstools," and "green pecans."

TABLE V. REGIONAL RATES PER 100,000 FOR ACCIDENTAL POISONING OF CHILDREN UNDER 5 YEARS, MOST COMMON SUBSTANCES: UNITED STATES, 1949-1950

SUBSTANCE	12 SOUTHERN STATES*	REMAINDER OF U. S.	TOTAL U. S.
Total	**4.09**	**2.00**	**2.58**
Barbiturates	0.09	0.10	0.10
Aspirin and salicylates	0.38	0.34	0.35
Petroleum products	1.41	0.35	0.65
Corrosives	0.45	0.08	0.19
Lead	0.04	0.27	0.20
Arsenic, antimony, etc.	0.32	0.05	0.13

*Alabama, Arkansas, Florida, Georgia, Louisiana, Mississippi, North Carolina, Oklahoma, South Carolina, Tennessee, Texas, Virginia.

UNCLASSIFIED

In the two years 40 cases were unclassifiable because of insufficient data on the death certificate.

STATE AND REGIONAL RATES

Numbers of deaths from poisoning occurring in some states are so few that their rates are not statistically reliable. One or two deaths per year may change the rate of a small state materially and give the erroneous impression of a good or bad situation. With stress on this caution, state ranking by rates is recorded in Chart II. The range is from 0 for Delaware (a small state which happened to have no deaths in the two-year period) to 6.2 for Mississippi.

Such a ranking reveals regional differences. With a few exceptions the highest rates were in southern states. Deaths occurred disproportionately often in twelve contiguous southern

states (Alabama, Arkansas, Florida, Georgia, Louisiana, Mississippi, North Carolina, Oklahoma, South Carolina, Tennessee, Texas, and Virginia). The rate for those states as a group was 4.09 per 100,000 as against a rate of only 2.00 for the remainder of the country (Table V).

In looking at the cause components of these rates, it is seen that for the barbiturates and aspirin there is little regional difference, but for petroleum products the southern states' rate is four times the rest of the country, and for corrosives and arsenic, about six times. Lead, curiously, is more com-

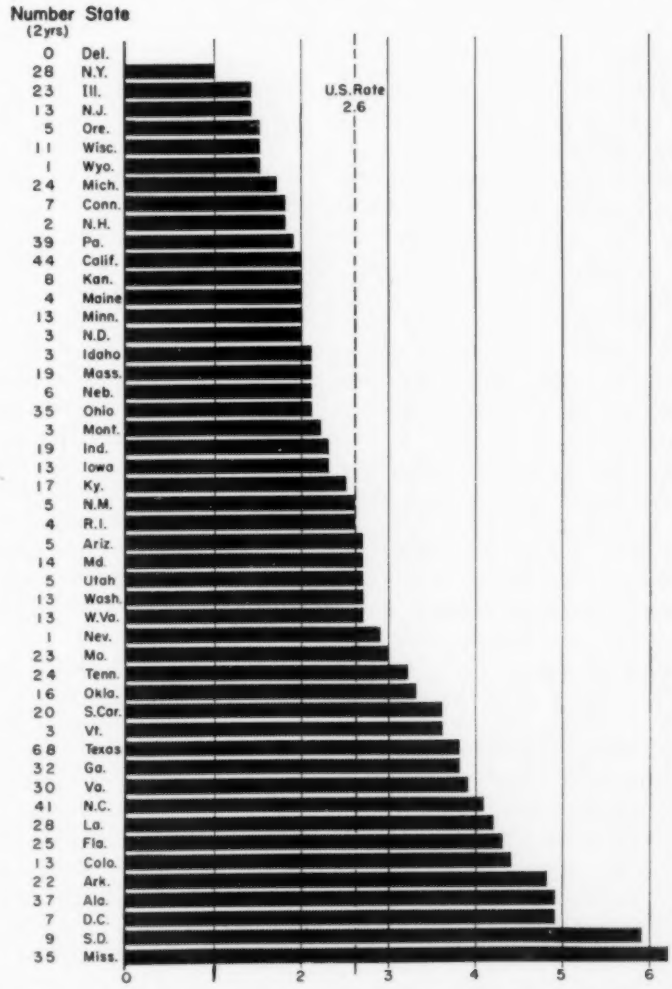

Chart II.—Number of deaths and annual death rate per 100,000 for accidental poisoning of children under 5 years: United States and each state, 1949 and 1950.

mon outside the South, perhaps reflecting regional differences in the use of paint or the greater availability of diagnostic laboratories in the urban centers.

DISCUSSION

The reasons behind the high United States rates for accidental poisoning are not entirely clear. A United States death rate from a specific cause four times that of a country with generally comparable over-all rates needs careful scrutiny. Easy access to poisonous substances is probably the basic reason. Our relatively high living standards make it possible for most family medicine cabinets to contain old prescriptions and over-the-counter medicines to which young children have easy access.

In 1951, the American people spent about three-quarters of a billion dollars on prescriptions and almost one billion on packaged medication, of which 135 million went for aspirin and analgesics.[3] The relationship of medicines in the home to income seems to be reflected in the higher rate for drug poisonings among white than among nonwhite children—0.9 as compared with 0.6. Craig and Fraser[4] in England have some evidence that medicinal poisonings increased at the time of the introduction of the National Health Service which increased the rate of prescribing.

On the other hand, many of the poisonings from material for external use probably have the related cause of poor housing and low income. Nonwhite rates from this cause are four times the white rates, 2.8 as compared with 0.7 (Table IV). Lye, kerosene, and rat poison are more commonly found in low-income homes. Lead poisoning has been definitely associated with poor housing.[5]

Roughly two-thirds of the deaths from accidental poisoning could be wiped out if aspirin, the barbiturates, kerosene, lye, lead, and arsenic were unavailable to small children. The physician's responsibility, especially in relation to drugs, is clear. It is probable that many physicians are on the alert to warn mothers about the dangers of rat poison or the new insecticides but are unaware of the danger of the aspirin bottle left carelessly lying around.

For each child dying of accidental poisoning there are many who are poisoned but recover. Poisoning, unlike many other types of accidents, does not produce a large number of cripples, except in the case of two substances, lye and lead. Esophageal stricture due to lye ingestion is a very common condition in the South, and lead encephalitis is well known in centers alerted and equipped to look for it.

Not all the drug deaths are due to accidental ingestion by an unsupervised runabout. Some, especially under the age of 1 year, must be due to wrong dosage. More information on the circumstances surrounding the accident is needed to point up precautionary measures. Each state might well have a committee of pediatricians who obtain data on each poisoning death and determine responsibility, as an educational and not punitive measure, as has been done so successfully for maternal deaths.

The lack of sufficient data for classification on 40 deaths in the two-year period points to the need for education of physicians as well as the public

in this problem. It is perhaps fair to assume that the quality of care rendered the poisoning victim by a physician who gives as the cause of death "pill," "tablet," "poison," or "dose of medicine" is not the highest. Or perhaps he is unaware of the value of obtaining an autopsy on an obscure case so that the accurate diagnosis can be used as a tool for prevention of a similar accident.

SUMMARY

1. Over 400 deaths of children under the age of 5 years occur each year in the United States from accidental poisoning.

2. The United States rate from this cause is four times the British rate.

3. Males outnumber females 3:2.

4. The nonwhite death rate is three times the white rate.

5. Drugs account for about one-third of the deaths, petroleum products, especially kerosene, for one-fourth, and material for external use for another one-third.

6. Aspirin, the barbiturates, kerosene, lye, lead and arsenic (and related compounds) account for about two-thirds of all accidental poisoning deaths in the under-5-year group.

7. State ranking by rates is shown, with caution expressed for use of rates based in some cases on such small numbers.

8. Twelve southern states have a rate twice that of the remainder of the United States, accounted for largely by kerosene and lye.

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THE PRESENT STATUS OF IDIOPATHIC EPILEPSY

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IDIOPATHIC or genetic epilepsy like cancer, diabetes, hemophilia, the muscular dystrophies, and other diseases still presents an enigma in medicine. All of these diseases have a number of features in common. They are diseases of antiquity; they are transmitted through heredity, and in the advanced stages they are incurable. Untreated, they usually follow a relentless, progressive course. The most intensive treatment, however effective, does not change nor remove the propensity of transmission of the disease to progeny. The transmission of hemophilia and most of the muscular dystrophies is too obvious to allow equivocation. The intelligent and educated public is perfectly aware of the hereditary factor in diabetes and in epilepsy. Why then do some physicians split infinitives to offer the unfortunate victims of epilepsy and their relatives false information about the genetics or hereditary transmission of the disease? Why encourage them to assume unnecessary responsibilities and serious hazards? I would not raise the issue of heredity except to reassure you that what you learned in your medical schools and from your textbooks is still basic and sound. Dissenting views in the public press may well be viewed with suspicion. Maudlin sympathy does not help the patient or the profession. Our present laws and regulations are based on centuries of experience. The physician with epilepsy in his family is not a qualified judge of their validity.

In the first monograph on epilepsy, written by a physician—Hippocrates, 400 B.C.—epilepsy is described as an hereditary disease.[1] In the last article by Lennox[2] in the *J. A. M. A.*, Aug. 29, 1953, the following comment is made:

"At this section meeting three years ago one of us presented electroencephalograms and other data on 122 twins subject to seizures. The series now numbers 155 pairs. *That a genetic factor (probably recessive in type) is important cannot be questioned,** but five other considerations must also be weighed: 1. The degree of heredity for epilepties varies widely. The average epileptic, if such existed, could expect that the chance for seizures in any given child would be about 1 in 30, but there would be a 50-50 chance that these would be limited to a few in childhood. Hence, likelihood of recurrent attacks would be perhaps only 1 in 60, or three times the chances of the average nonepileptic. A few of these cases, the most serious, would be the direct result of some acquired brain disease. 2. Epilepsy per se is not inherited, and only *the tendency is.** Hence carriers of the tendency, many times more numerous, are genetically much more important than the affected. Unless carriers are controlled, even sterilization of all epi-

*Italics mine.

lepties would not appreciably reduce the incidence for many generations. 3. Possession by parents of transmissible mental and physical traits that are desirable may outweigh an undesired tendency to seizures on the part of one parent. 4. A transmitted predisposition, what Galen called 'an aptitude of the body,' *is no more important for epilepsy than for many other diseases.** Progeny denied for one should be denied for all. 5. Methods of prevention and control of acquired epilepsy are at hand. Prevention of brain disease and also successful drug treatment have advanced importantly in recent years, and additional gains are probable.

"Certainly persons with strong evidence of heredity of seizures and of poor mental or physical traits should not procreate. . . ."*

What difference does it make to the epileptic whether he inherited the disease or a "tendency to the disease"? Once the disease has been diagnosed and established, treatment and supervision must be continued indefinitely. And even though the disease is completely controlled, the tendency is transmitted just the same. This does not imply that the patient should not be adequately treated and encouraged.

From the monograph by Hippocrates, we may skip to the one by Gowers[3] (1901) in which is stated the following:

"We may safely conclude that in at least 50 per cent of all cases of epilepsy the malady is ultimately the result of neurotic inheritance. The proportion may be much larger, but its extent is not at present to be discerned."

In one of the latest monographs devoted to "Epilepsy and Convulsive Disorders in Children" by Bridge[4] (1949), the author makes the following statements:

"The most significant material has been presented by Brain,[5]* Lennox and Cobb,[6] Stein,[7] Himler,[8] Conrad,[9] and Lennox.[10] The data suggests that convulsive disorders are several times more prevalent among the relatives of epilepties than among those of nonepileptic individuals. Although the common errors in carrying out such studies tend to give an exaggerated difference between the two groups, the fact appears indisputable from a statistical point of view.

"If heredity plays a significant role in the causation of epilepsy, seizures should be found more frequently among near relatives of epileptic patients than among more distant ones. Several investigations demonstrate this to be the case. Conrad[9] has made a detailed review of the subject and concludes with the figures presented in . . .

"Since 1939, Lennox, Gibbs, and Gibbs[11, 14] have called attention to the fact that abnormal waves might be detected in the electroencephalogram in a significant proportion of supposedly normal people. Other workers have since confirmed this observation. The findings are more common among relatives of epileptic than of nonepileptic individuals. As a result of a large series of observations these authors conclude that approximately 15 per cent of the entire population shows some abnormality in brain waves. They advance the hypothesis that cerebral dysrhythmia (epilepsy) is an inherited condition, possibly a dominant trait. The 10 to 15 per cent of the g e n e r a l population that has dysrhythmia without seizures represents latent epilepsy. Whether or not convulsive symptoms develop in such a setting depends on the occurrence of other factors, notably injuries causing the formation of scars in the brain. Viewed in this way heredity assumes great importance in the causation of epilepsy, and no family can feel secure

*Italics mine.

*Numbers within quoted material refer to references in original publication.

in having children without first subjecting themselves to electroencephalographic studies.

"From the foregoing discussion there can be little doubt that hereditary factors contribute to the development of epilepsy in certain individuals. In others there still remains the possibility that some added degree of susceptibility has been acquired through family traits, although this element appears to be less prominent as a cause of the disease."

Ounsted[5] reported the following in December, 1952:

"Modern authors have found that a constitutional tendency to suffer from fits is evident not only in epilepsy but also in febrile convulsions (Lennox, 1949), eclampsia (Rosenbaum and Maltby, 1943), anaesthetic convulsions (William and Sweet, 1944), convulsions following minor brain injury (Bridge, 1949).

"The emphasis placed on the etiological force of the inherited factor varies widely. This appears to be due in part to selective sampling. Thus, some studies are based on epilepties in institutions, others on epilepties with associated mental deficiency, others on series of dizygous and monozygous twins, and others on patients whose seizures are provoked by fever.

"We are trying to learn something about the natural history of convulsive disorders of children in Oxford, and, to that end, we are collecting a fresh series of consecutive cases. The material presented here is a pilot analysis of the family histories of the first 337 children entering the sample.

"Of the 333 propositi with whole or half trees, 130 had a kinsman with a clear history of a convulsive disorder. Thus, the incidence of children with positive family histories was 39 per cent."

Kimball[6] in March, 1942, reported the results of his study at a special school clinic in Detroit:

"In 578 cases in which the history was known, epilepsy occurs in the direct lineage in 375 cases or 64.9 per cent. The diagnosis in 1250 cases was listed as Hereditary epilepsy in 35.1 per cent; Epilepsy, heredity unknown in 39.9 per cent; Brain injury or defect in 12.2 per cent; idiopathic epilepsy in 8.6 per cent; chorea in 2 per cent; questionable diagnosis in 4 per cent."

My own experience confirms the conclusions of those who have made thorough studies on adequate material for a sufficiently long time. The longer one is able to follow his cases the more evidence of heredity he finds. In my series of cases, over 50 per cent of the patients with genetic epilepsy give a history of epilepsy in the family.

DIAGNOSIS

With the introduction of the electroencelphalograph by Berger[7] in 1929, it became possible for the first time in medical history to confirm or establish the diagnosis of epilepsy and to distinguish the various types.[8] While all of the various forms and manifestations of the disease are components of the same entity, they vary greatly in their clinical forms and in their response to treatment. This makes the electrogram of inestimable value: first, in confirming the diagnosis of epilepsy with objective evidence and in establishing the type or types; second, in offering means for determining the carrier in the parents and/or relatives and the potential in siblings; third, the electrogram offers means for evaluation of the results of treatment with a realization, of course, of definite limitations. A negative electrogram never outweighs a clinical diagnosis based on a careful study. A number of electrograms may be necessary to obtain a satisfactory tracing. The interpretation of the electrogram must

be carefully made and guarded and allows no flight of imagination nor basis of treatment. As with the electrocardiogram or the roentgenogram, the electroencephalogram is a diagnostic aid only.

However, with this apparatus, the known clinical types of epilepsy, such as grand mal and petit mal, have been established. The "abnormal mental states or psychic equivalents"[9] have been identified as "psychomotor" or "temporal lobe" syndromes.[10] Akinetic seizures have been identified as probable residuals of brain damage and some of the behavior disturbances of childhood have been given a new interpretation.[11]

TREATMENT

A great deal of time and study has been given to new anticonvulsants. As a result we now have phenacemide, the hydantoins, the diones, and other new drugs at our disposal.[12] However, experience proves that phenobarbital still is the most effective, the most reliable, and the least toxic anticonvulsant for the treatment of grand mal and major convulsions. It has absolutely no effect on petit mal seizures. It may precipitate behavior outbursts in psychomotor or temporal lobe epilepsy or in chronic encephalitis. It is thus useful as a diagnostic-therapeutic agent.

Metharbutal or Gemonil is a recently added barbiturate which is a second choice after phenobarbital. It is most effective in major convulsions but may modify akinetic seizures and psychomotor and temporal lobe epilepsy.

Phenacemide (Phenurone) is the first drug we have had which helps to control some of the psychomotor and temporal lobe epilepsies.[11, 12]

The diones, Tridione and Paradione, are the first and only drugs to control about one-third of the petit mal seizures. Milontin has not been effective in my hands.

The hydantoins available at present are limited to Dilantin (sodium phenytoin) and Mesantoin (methyl-phenyl-ethyl hydantoin). The hydantoins are undesirable drugs in children because the therapeutic and the toxic levels are too close. The side effects are too disagreeable for a drug which has a low degree of effectiveness. Phenacemide, the diones, and the hydantoins are toxic drugs to be used only under close supervision with blood counts and liver tests. Nuvarone (3-methyl-5-phenyl-hydantoin) is an effective hydantoin with little toxicity.*

Themisone (atrolactamide) is a new drug which has been reported to be effective in the control of some resistant major seizures.[13]

Mysoline (5-phenyl-5-ethyl-hexahydropyrimidine-4-6-dione) has also been reported to be effective in the control of major convulsions.[14] The side reactions are disturbing.

Aureomycin is reported to control a number of resistant cases of epilepsy and to even correct the abnormalities in the electrogram.[15] I have tried Aureomycin in large doses in eighteen children with severe, persistant, and resistant convulsions, with no effect. It is possible that the drug has an anticonvulsant effect on some patients.

Ammonium chloride[16] and caffeine[17] were discarded long before they were rediscovered as "new drugs."[18, 19] I have found Hibicon effective in only two of sixty-eight patients. Glutamic acid has not survived the test of time.

*Report in preparation.

The ketogenic diet[20] remains the most effective treatment available and the only treatment for all forms of the disease and aimed at a correction of the basic disorder. However, the diet requires a preliminary fast of ten to fourteen days and rigid supervision of the food intake.

Intensive prolonged narcosis produced with diphenylhydantoin[21] is drastic at best, to be tried only in hopeless adults.

The objective of treatment is, of course, the complete control of convulsions. Only then can the patient and his parents relax and establish a feeling of security and confidence. This goal is difficult to attain and is possible in only about half of the patients. Claims of better results with new drugs are usually not subject to verification. Results should be classified as complete control or freedom from seizures, good control, improvement, and failure. A reduction of seizures which does not allow normal activity and freedom from supervision is of limited value.

If the drug selected is not completely effective in one month, another must be substituted and, if necessary, the entire list must be tried. If the patient is still not completely controlled, combinations must next be used. The first choice in major convulsions is phenobarbital or metharbutal with an hydantoin. The combined drugs in one tablet or capsule do not allow proper regulation of individual drug dosage.

Phenacemide should not be given with the hydantoins nor with the diones. Two hydantoins should not be given together. The hydantoins, the diones, and Phenurone are toxic drugs to be used only when necessary and under close supervision with blood counts. However, persistent, uncontrollable convulsions may require drastic treatment.

The drug of choice should be administered in increasing doses to effectiveness every six or eight hours or in enteric-coated tablets or capsules every twelve hours. Once the convulsions are under control, beyond the expected time of recurrence, drugs should be continued at least one year and, if possible, until a normal electrogram is obtained. They may then be tapered off gradually for another year or two. No anticonvulsant should be stopped abruptly, even if replaced.

CONCLUSIONS

A brief summary of the present status of idiopathic or genetic epilepsy is presented. It is based on an experience of two years of treatment at the Mayo Clinic followed by twenty-eight years in private and hospital practice in Milwaukee. The hereditary transmission of the disease is established. The value of the electroencephalogram is presented. The useful drugs are listed.

I do not wish to sound pessimistic about the future for the epileptic patient, and I feel that he should be given all the encouragement possible. However, I do believe that the medical profession should not be given misinformation or patients false encouragement. There are no miracle men nor miracle drugs in epilepsy.

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AN EVALUATION OF LARGE DOSES OF CORTISONE IN FIRST ATTACKS OF RHEUMATIC CARDITIS

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THE beneficial effect of cortisone on the nonspecific or constitutional manifestations of rheumatic fever is well documented.[1, 2, 3, 4, 5] Fever, tachycardia, toxemia, and anorexia are influenced favorably. Euphoria, increased appetite, and weight gain are decided advantages of cortisone therapy. Certain abnormal laboratory findings, including elevated sedimentation rate and increased serum fibrinogen and globulin levels, tend to return to normal. The immediate suppressive effect of cortisone in controlling the clinical manifestations of acute carditis is striking. However, there is considerable doubt as to whether cortisone prevents or significantly reduces residual damage to the heart muscle and valves.[6]

It is difficult to evaluate results of therapy in a disease like rheumatic fever, for which there is no specific diagnostic test, which is so variable in its course, and which is subject to spontaneous remissions and exacerbations. Despite these limitations, earlier studies suggested that results were better when treatment was early and adequate.[7, 8, 9, 10, 11, 12]

A recent study[13] casts doubt on "uncontrolled" observations and concludes that cortisone and adrenocorticotropic hormone are no better than salicylates in their immediate effect on rheumatic fever and on carditis. It is our purpose to present additional clinical data to lend support to the contention that *early, intensive,* and *individualized* cortisone therapy may prevent residual cardiac damage in first attacks of rheumatic carditis.

MATERIAL AND PLAN OF TREATMENT

Since 1951, we have treated and followed a large series of patients with acute and chronic rheumatic fever. This report represents the clinical data of *only* those patients in their first attack of acute carditis. The remaining patients were either cases of chronic smoldering activity or recurrent rheumatic fever. The former were not treated with cortisone. The results with cortisone in the recurrent cases are the subject of another report.

The material to be presented includes nineteen children between 5 and 15 years of age, all of whom suffered from their first attacks of rheumatic fever with carditis, and all of whom were treated with cortisone, in dosage and duration differing from that of the International Cooperative Study. Early in our work it became apparent that it was extremely difficult to select patients with rheumatic fever and

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carditis of comparable symptomatology, severity, and duration, and to treat half with salicylates and half with cortisone. Thus all our patients received cortisone but in higher dosage than reported in the literature. The other variable over which we had no control was the duration of illness before therapy. The effect of *higher* dosage in early and late cases is to be derived from our material.

The diagnosis of rheumatic fever was made according to the criteria of Jones, as modified by Denny and associates.[14] Two major, or one major and two minor, manifestations of the disease were always present. Our criteria for the diagnosis of carditis were most critical and included (1) a new harsh systolic murmur at the apex transmitted into the left axilla, (2) a new diastolic murmur either at the apex, aortic area, or Erb's point, (3) pericardial effusion, or a friction rub, (4) unmistakable cardiac enlargement in a first attack of rheumatic fever, determined by fluoroscopy and x-ray, (5) electrocardiographic changes, and (6) "overactive" heart, muffled quality of heart sounds, gallop rhythm, and a disproportionate tachycardia.

All patients in this series were at complete bed rest. The dietary sodium was restricted to 200 to 500 mg. a day. The caloric intake was adequate in that it maintained or increased the body weight. Fluid intake was not restricted. Daily body weight and fluid intake and output were recorded. For the first three hospital days, 300,000 units of procaine penicillin was administered intramuscularly to clear the throat of beta hemolytic streptococci. Two hundred thousand units of oral penicillin was then administered daily for prophylaxis against streptococcal infection. The initial total daily dose of cortisone was between 200 and 300 mg., given in four divided oral doses. This dose was usually continued for at least two weeks or even longer, until the clinical and laboratory findings indicated maximal suppression of the disease. It was only then that the dose was reduced gradually. If symptoms or signs recurred, the higher dosage was resumed. On the average, 200 to 300 mg. of cortisone was given for two weeks, 150 mg. for 1 to 2 weeks, 100 mg. for one week, and 50 mg. for one week. Dosage was individualized according to the severity of the disease and its course.

All patients were examined frequently by the same physicians. X-ray and fluoroscopy were done at frequent intervals. Electrocardiograms (twelve leads) were recorded at approximately weekly intervals. Complete blood counts were done weekly. The chemical examination of the blood included determinations for sugar, urea nitrogen, sodium, potassium, chloride, CO_2 content, total protein, albumin, and globulin. Laboratory tests for rheumatic activity included the blood sedimentation rate, fibrinogen, and precipitation tests for abnormal protein concentrations—cephalin flocculation, thymol turbidity, gamma globulin, and zinc turbidity. These chemical determinations were done weekly.

The total care program of the rheumatic fever ward at the Jewish Sanitarium and Hospital for Chronic Diseases permitted continued observation of all patients. The follow-up study was made in the Cardiac Clinic of the Jewish Sanitarium and Hospital for Chronic Diseases and the Pediatric Cardiac Clinic of the Jewish Hospital of Brooklyn.

It should be stressed that the evaluation of cortisone therapy is based on clinical observation and complete absence of any manifestation of heart disease after a follow-up period of six to fourteen months.

CASE ABSTRACTS

CASE 1.—J. M., a 7-year-old white boy, was admitted to the hospital because of fever, polyarthritis, and carditis. Examination of the heart revealed a moderately loud, harsh apical systolic murmur transmitted to the axilla. Laboratory findings included an enlarged heart by fluoroscopy and x-ray, a prolonged P-R interval, an elevated sedimentation rate, a reversal of the serum albumin/globulin ratio, and anemia. Cortisone was started on the sixth day of illness. The patient received 3.7 Gm. in forty-two days. After therapy all his symptoms had cleared and only a Grade 2 systolic murmur was heard at the pulmonic area. Four months later, even this murmur had disappeared. Eight months later, there were no cardiac abnormalities.

CASE 2.—H. C., a 12-year-old white boy, was admitted to the hospital because of polyarthritis, fever, lower abdominal pain, lethargy, and carditis. Examination of the heart revealed a moderately loud, harsh apical systolic murmur transmitted to the axilla. Laboratory findings included an elevated sedimentation rate, leukocytosis, and a prolonged P-R interval. Fluoroscopy and x-ray showed no enlargement of the heart. Cortisone was started six days after the onset of symptoms. He received 3.9 Gm. in thirty-seven days, with complete subsidence of all symptoms. Complete cardiac examination two months later revealed only a soft systolic murmur localized to the pulmonic area. This murmur disappeared within eight months.

CASE 3.—B. J., a 12-year-old white boy, was admitted to the hospital because of fever, polyarthritis, fatigability, and carditis. Examination of the heart revealed a moderately loud, harsh apical systolic murmur transmitted to the axilla and along the left sternal border. Laboratory findings included a prolonged P-R interval, reversal of the serum albumin/globulin ratio, elevated sedimentation rate, anemia, and leukocytosis. Cortisone was started on the sixth day of illness. The patient received 3.9 Gm. in forty-one days, with complete recovery at the conclusion of therapy. Six months later, there were no cardiac abnormalities.

CASE 4.—I. C., a 10-year-old white boy, was admitted to the hospital because of polyarthritis, fever, substernal pain, dyspnea, and carditis. Examination of the heart revealed enlargement to the right and left, muffled heart sounds, tachycardia, a to-and-fro pericardial friction rub, and a moderately loud, harsh apical systolic murmur transmitted to the axilla. The x-ray was characteristic of pericarditis and pleuritis. Laboratory findings included a very high sedimentation rate, leukocytosis, a prolonged P-R interval, and reversal of the serum albumin/globulin ratio. Cortisone was started on the eighth day of illness. The patient received 6.7 Gm. in fifty-four days, with complete subsidence of all symptoms, and with only a soft systolic murmur persisting at the pulmonic area. One month later, he had a recurrence of polyarthritis and carditis, which was treated from the first day of illness with ACTH for twenty-six days, with complete recovery. Fourteen months later there were no cardiac abnormalities.

CASE 5.—P. K., an 8-year-old white boy, was admitted to the hospital because of fever, fatigability, polyarthritis, and carditis. Examination of the heart revealed premature contractions and a moderately loud, harsh apical systolic murmur transmitted to the axilla. Laboratory findings included a high sedimentation rate, anemia, a prolonged P-R interval, and reversal

of the serum albumin/globulin ratio. Cortisone was started on the eighth day of illness. The patient received 2 Gm. in twenty days with complete recovery. A soft systolic murmur heard at the pulmonic area had disappeared six months later.

CASE 6.—E. W., a 13-year-old white girl, was admitted to the hospital because of fever, polyarthritis, and carditis. Examination of the heart revealed a moderately loud, harsh apical systolic murmur transmitted to the axilla and to the left base. Laboratory findings included an elevated sedimentation rate, a prolonged P-R interval, anemia, and reversal of the serum albumin/globulin ratio. Cortisone was started on the eighth day of illness. The patient received 4.1 Gm. in thirty-seven days, with complete recovery. Six months later, there were no cardiac abnormalities.

CASE 7.—D. B., a 5-year-old Negro boy, was admitted to the hospital because of fever, polyarthritis, abdominal pain, dyspnea, and carditis. Examination of the heart revealed muffled heart sounds, frequent premature contractions, and a moderately loud, harsh apical systolic murmur transmitted to the axilla and base. Fluoroscopy and x-ray revealed the heart enlarged to the right and left. Laboratory findings included an elevated sedimentation rate, a prolonged P-R interval, reversal of the serum albumin/globulin ratio, anemia, and leukocytosis. Cortisone was started on the tenth day of illness. The patient received 3.6 Gm. in thirty-eight days, with complete recovery. A soft systolic murmur was heard at the pulmonic area. One month later, even this murmur had disappeared. Ten months later there were no cardiac abnormalities.

CASE 8.—G. M., an 11-year-old white boy was admitted to the hospital because of fever, polyarthritis, and carditis. Examination of the heart revealed a moderately loud, harsh apical systolic murmur transmitted to the axilla, and a persistent tachycardia in the absence of fever. Laboratory findings included a prolonged P-R interval, and an elevated sedimentation rate. Cortisone was started on the eleventh day of illness. The patient received 8.9 Gm. in forty-five days. At the end of treatment, a faint, soft systolic murmur was heard at the apex. Five months later, even this murmur had disappeared. Twelve months later, there were no cardiac abnormalities.

CASE 9.—P. G., a 6-year-old white girl, was admitted to the hospital because of fever, polyarthritis, and carditis. Examination of the heart revealed tachycardia in the absence of fever, a moderately loud, harsh apical systolic murmur transmitted to the axilla and to the left base, and a decrescendo, blowing diastolic murmur at Erb's area. Laboratory findings included an elevated sedimentation rate, an enlarged heart by fluoroscopy and x-ray, and a reversal of the serum albumin/globulin ratio. Cortisone was started on the fifteenth day of illness. The patient received 6.5 Gm. in forty-five days. The diastolic murmur at Erb's area disappeared. At the end of treatment, a faint systolic blow persisted at the apex. Three months later this murmur disappeared and no cardiac enlargement was revealed by fluoroscopy and x-ray. Eleven months later there were no cardiac abnormalities.

CASE 10.—M. R., a 14-year-old white girl, was admitted to the hospital because of fever and a moderately severe chorea. She was very irritable, and unable to button her clothes or to stand. Examination of the heart revealed a moderate tachycardia, but no murmurs. However, two days later, a loud, harsh apical systolic murmur with transmission to the axilla was heard. Laboratory findings included a markedly elevated sedimentation rate, leukocytosis, and a reversal of the serum albumin/globulin ratio. Cortisone was started on the twenty-

first day of chorea, but only two days after the appearance of the apical murmur. The patient received 3.7 Gm. of cortisone in thirty-eight days. After one week of therapy, the coarse choreiform movements and the emotional lability had cleared. The remaining choreiform movements cleared during the second week of treatment. By the end of treatment, the murmur had disappeared and the heart was not shown to be enlarged by fluoroscopy and x-ray. Eight months later there were no cardiac abnormalities.

CASE 11.—R. J., an 8-year-old white boy, was admitted to the hospital because of fever, polyarthritis, lethargy, and carditis. Examination of the heart revealed a marked tachycardia, gallop rhythm, a moderately loud, harsh apical systolic murmur transmitted to the axilla and left base, and a soft low-pitched mid-diastolic murmur at the apex. Laboratory findings included a markedly elevated sedimentation rate, an enlarged heart by fluoroscopy and x-ray, a prolonged P-R interval, a reversal of the serum albumin/globulin ratio, anemia, and leukocytosis. Cortisone was started on the twenty-eighth day of illness. Six grams was administered in fifty-one days. By the end of treatment, a soft systolic murmur at the pulmonic area could be heard. This murmur disappeared within two weeks. Nine months later there were no cardiac abnormalities.

CASE 12.—M. M., an 11-year-old white girl, was admitted to the hospital because of fever, polyarthritis, and carditis. Examination of the heart revealed gallop rhythm, muffled heart sounds, and a moderately loud, harsh apical systolic murmur transmitted to the axilla. Laboratory findings included an elevated sedimentation rate, an enlarged heart by fluoroscopy and x-ray, a prolonged P-R interval, and a reversal of the serum albumin/globulin ratio. Cortisone was started on the twenty-fourth day of illness. The patient was given 6.3 Gm. in fifty days. By the end of treatment, a faint systolic murmur was still present at the apex and pulmonic area. Three months later, only a soft systolic murmur at the pulmonic area could be heard. Eight months later this faint murmur was still audible, but there was no cardiac enlargement.

CASE 13.—R. P., a 13-year-old white boy, was admitted to the hospital because of fever, polyarthritis, and carditis. Examination of the heart revealed a pericardial friction rub, and a moderately loud, harsh apical systolic murmur transmitted to the axilla and the pulmonic area. Laboratory findings included an elevated sedimentation rate, an enlarged heart by fluoroscopy and x-ray, a prolonged P-R interval, a reversal of the serum albumin/globulin ratio, anemia, and leukocytosis. Cortisone was started on the sixth day of illness. The patient received 5.0 Gm. in sixty days. A faint systolic murmur at the apex persisted at the end of treatment, and was still present three months later. This murmur disappeared within twelve months.

CASE 14.—M. M., a 6-year-old white boy, was admitted to the hospital because of fever, marked dyspnea, and carditis. Examination of the heart revealed marked tachycardia, gallop rhythm, and a very loud, harsh apical systolic murmur transmitted to the axilla. A few fine moist râles were heard at the right lung base. Laboratory findings included a markedly enlarged heart by fluoroscopy and x-ray, a prolonged P-R interval, an elevated sedimentation rate, a reversal of the serum albumin/globulin ratio, and anemia. Cortisone was started on the twenty-first day of illness. Prompt relief of the carditis and heart failure followed. The patient received 5.4 Gm. in forty-five days. At the end of treatment a loud apical systolic murmur persisted. He continued to have low-grade fever, tachycardia, and an elevated sedimentation rate for ten months after cortisone therapy had been discontinued. Re-examination twenty-four months after cortisone

therapy revealed inactive rheumatic heart disease with mitral insufficiency.

CASE 15.—C. J., a 6-year-old white girl, was admitted to the hospital because of fever, marked dyspnea, orthopnea, and carditis. Examination of the heart revealed enlargement by fluoroscopy and x-ray, gallop rhythm, marked tachycardia in the absence of fever, and a loud, harsh apical systolic murmur transmitted to the axilla and left base. There were fine moist râles at both bases. Laboratory findings included broad P waves in Leads I and II, isoelectric T waves in Lead I, an elevated sedimentation rate, reversal of the serum albumin/globulin ratio, anemia, and leukocytosis. Cortisone was started on the twenty-fifth day of illness. The patient received 5.2 Gm. in forty-one days. The immediate effect of cortisone on the congestive failure was prompt. However, low-grade fever, tachycardia, and elevation of the sedimentation rate continued for six months after cortisone had been discontinued. Re-examination seventeen months after cortisone therapy revealed inactive rheumatic heart disease with mitral insufficiency.

CASE 16.—M. T., a 15-year-old Negro boy, was admitted to the hospital because of fever, polyarthritis, and carditis. Examination of the heart revealed a moderately loud, harsh apical systolic murmur transmitted to the axilla and left base. Laboratory findings included an elevated sedimentation rate, an enlarged heart by fluoroscopy and x-ray, a prolonged P-R interval and occasional auricular premature contractions, and a reversal of the serum albumin/globulin ratio. Cortisone was started on the thirty-fifth day of illness. The patient received 5.4 Gm. in forty-four days. Despite therapy the apical systolic murmur was still audible fourteen months after treatment.

CASE 17.—S. N., a 10-year-old white girl, was admitted to the hospital because of fever, polyarthritis, carditis, and severe epistaxis. Examination of the heart revealed a loud, harsh apical systolic murmur transmitted to the axilla. Laboratory findings included elevated sedimentation rate, a prolonged P-R interval, and reversal of the serum albumin/globulin ratio. Cortisone was started on the fifty-sixth day of illness. The patient received 5.3 Gm. in forty-two days. Following therapy the murmur at the apex was still present but was less intense. Examination two months later revealed a murmur typical of mitral insufficiency. The same murmur was audible nine months later.

CASE 18.—A. B., an 11-year-old white girl, was admitted to the hospital because of fever, recurring polyarthritis, abdominal pain, and carditis. Examination of the heart revealed a moderately loud, harsh apical systolic murmur transmitted to the axilla. Laboratory findings included a prolonged P-R interval, an elevated sedimentation rate, reversal of the serum albumin/globulin ratio, and anemia. Cortisone was started after about five months of illness. The patient received 9.7 Gm. in sixty-two days. Low-grade rheumatic activity continued for two months after cortisone therapy. For the next ten months, although all activity had subsided, an apical systolic murmur persisted.

CASE 19.—M. J., a 9-year-old white boy, was admitted to the hospital because of fever, polyarthritis, and carditis. Examination of the heart revealed marked enlargement, gallop rhythm, and a harsh, loud apical systolic murmur transmitted to the axilla. Laboratory findings included a prolonged P-R interval, reversal of the serum albumin/globulin ratio, and anemia. Cortisone was started on the fifty-sixth day of illness. The patient received 2.5 Gm. in twenty-two days. The day following the cessation of cortisone therapy, there was recurrence of fever, leukocytosis, and severe abdominal pain, but the sedimentation rate remained normal. A diagnosis of appendicitis was considered, but he

was treated only with large doses of antibiotics. Within three days, the symptoms subsided. He continued to have low-grade fever, elevated sedimentation rate, and tachycardia. Ten months after cortisone therapy, severe carditis developed characterized by severe chest pain, dyspnea, orthopnea, exacerbations of carditis which have been controlled by either cortisone or ACTH.

OBSERVATIONS

The summary of the observed signs and symptoms of rheumatic fever and carditis is incorporated in Table I. All nineteen patients had significant organic murmurs; eleven had cardiac enlargement as demonstrated by fluoroscopy and x-ray. In addition, fifteen patients had abnormal electrocardiographic findings, and three were in heart failure.

TABLE I

MAJOR MANIFESTATIONS		MINOR MANIFESTATIONS	
1. Carditis		1. Fever	19
a. Cardiac enlargement	11	2. Abdominal pain	1
b. New murmurs	19	3. Erythema marginatum	0
c. Friction rub	2	4. Epistaxis	1
d. Abnormal electrocardiogram	15	5. Elevated sedimentation rate	19
e. Cardiac failure	3		
2. Migratory polyarthritis	16		
3. History of recurrences	0		
4. Chorea	1		
5. Subcutaneous nodules	0		

TABLE II

CASE	AGE	SEX	COLOR	DAYS OF ILLNESS BEFORE TREATMENT	DAYS OF TREATMENT	TOTAL DOSE (GM.)	HEART BEFORE TREATMENT	HEART AFTER TREATMENT	FOLLOW-UP (MONTHS)
1. J. M.	7	M	W	6	42	3.7	MS	Normal	8
2. H. C.	12	M	W	6	37	3.9	MS	Normal	8
3. B. J.	12	M	W	6	41	3.9	MS	Normal	6
4. I. C.	10	M	W	8	54	6.7	MS, FR	Normal	14
5. P. K.	8	M	W	8	20	2.0	MS	Normal	6
6. E. W.	13	F	W	8	37	4.1	MS	Normal	6
7. D. B.	5	M	N	10	38	3.6	MS	Normal	10
8. G. M.	11	M	W	11	45	8.9	MS	Normal	12
9. P. G.	6	F	W	15	45	6.5	MS, AD	Normal	11
10. M. R.	14	F	W	21	38	3.7	MS	Normal	8
11. R. J.	8	M	W	28	51	6.0	MS, MD	Normal	9
12. M. M.	11	F	W	24	50	6.3	MS, G	PS±	8
13. R. P.	13	M	W	6	60	5.0	MS, FR	Normal	12
14. M. M.	6	M	W	21	45	5.4	MS, G	MS	24
15. C. J.	6	F	W	25	41	5.2	MS, G	MS	17
16. M. T.	15	M	N	35	44	5.4	MS	MS	14
17. S. N.	10	F	W	56	42	5.3	MS	MS	9
18. A. B.	11	F	W	5 mo.	62	9.7	MS	MS	12
19. M. J.	9	M	W	56	26	2.5	MS, G	MS	25

Note: MS = mitral systolic, FR = friction rub, AD = aortic diastolic, MD = mitral diastolic, G = gallop rhythm, PS = pulmonic systolic.

Table II summarizes the clinical findings. There was no residual cardiac involvement in Cases 1 through 13. Of these thirteen patients, ten were treated within the first two weeks of their illness. It should be stressed that all thirteen patients suffered from monocyclic attacks of rheumatic fever, and that the disease was inactive at the end of cortisone therapy. Six patients had residual systolic murmurs after cortisone therapy. These patients were treated late in the disease, and either had a long, protracted low-grade activity, or a polycyclic course.

The anti-inflammatory response was similar to that already described in the literature. The temperature usually fell within two to three days. Polyarthritis usually subsided within three days. Although there was great variation, sedimentation rate usually returned to normal within two weeks and the increased serum globulin within three to four weeks. Euphoria and increase in appetite made the management of these sick children much easier. Salicylates did not have this advantage.

The immediate suppressive effect of cortisone on symptoms of carditis was remarkable. In three severely ill children (Cases 14, 15, 19) cortisone therapy was considered lifesaving. In one of these cases (Case 19) salicylates had been ineffective.

Gallop rhythm usually disappeared within four days, and tachycardia responded within a week, except in those cases where carditis was already of several weeks' duration before therapy was begun. In several patients, cortisone produced sinus bradycardia and arrhythmia with nodal escape. The prolonged P-R interval usually returned to normal within two weeks. The friction rub in Cases 4 and 13 disappeared within four days. No new murmurs developed in our series of cases. The intensity of a murmur increased at times during cortisone therapy. This was probably related to the slowing of the heart rate and increased stroke volume.

Case 19 was one of our first patients treated. We now feel he was inadequately treated despite the fact that he represents a patient with chronic low-grade activity punctuated with acute exacerbations.

Most patients showed some degree of "rebound" when cortisone therapy was stopped. Clinical and/or laboratory "rebound" included such signs and symptoms as fever, joint pains, tachycardia, and elevated sedimentation rate. At times, it was difficult to evaluate whether signs and symptoms represented "drug rebound" or continuing rheumatic activity. A spontaneous recovery without therapy within a week or two suggested "rebound." However, it must be admitted that this differentiation is arbitrary for want of a specific test for rheumatic activity.

Despite the high total dosage of cortisone employed, there were no complications. All patients developed the Cushing habitus, with moon-face, obesity, and striae. Mild facial hirsutism and acne never necessitated cessation of therapy. Elevation of the blood sugar and glycosuria were not encountered. A few skeletal surveys at the outset of our study failed to reveal demonstrable osteoporosis. Hepatomegaly encountered in the first week or two probably represented congestive failure induced by an endogenous water shift secondary to

cortisone therapy.[15] Hepatomegaly encountered later probably represented fatty infiltration of the liver.[16] In both instances, there was no contraindication to continued cortisone therapy.

DISCUSSION

The chief aim in cortisone therapy is to suppress the earlier exudative inflammatory response of rheumatic carditis so as to prevent and minimize the subsequent fibroplastic reaction and valvular deformity. Early in the course of our observations,[17, 18] it became apparent that an arbitrary fixed dosage schedule, such as employed by the International Cooperative Study, was at times insufficient to suppress obvious signs and symptoms of inflammation. Their schedule[19] of 300 mg. cortisone for one day followed by 200 mg. for four days, 100 mg. for three weeks, 75 mg. for the fourth and fifth weeks, and 50 mg. for the sixth week, was inadequate when the rheumatic fever was severe. It seemed logical to give larger doses until suppression of all apparent signs and symptoms of the disease was effected. Fortunately, the minimal side effects of large doses of cortisone in our series of children and adolescents permitted its continued use.

An analysis of our cases treated with individualized and large dosage clearly shows that the patient with a monocyclic attack of rheumatic fever has a better chance of escaping cardiac damage if adequately treated early in the disease. Conclusive statements on the basis of this small series of cases are not warranted, but the trend shown in this study warrants the use of larger doses of cortisone given early in the disease and continued for a long enough period. In a monocyclic attack, it may be possible in this way to prevent residual cardiac damage.

Our experience also indicates that cortisone in adequate dosage may be more effective in controlling carditis than salicylates. In this we agree with the observations of Bunim and associates.[20, 21] The dangers of salicylate intoxication are greater than the side effects we have observed with cortisone. The euphoria and increase in appetite with cortisone are salutary effects which are welcome in the treatment of the severely ill child with carditis, with or without congestive failure.

SUMMARY

1. Our experience in the treatment of nineteen patients in their first attacks of rheumatic fever with carditis is presented in this paper.

2. The analysis of our material tends to support the contention that early individualized treatment of a monocyclic attack of carditis with large doses of cortisone may prevent residual valvular damage.

3. Initial doses of 300 mg. and higher for a period of two weeks or more may be necessary for suppression of rheumatic activity in individual cases.

4. Cortisone may be more effective in controlling certain instances of carditis than salicylates.

5. The side effects of cortisone therapy in the children and adolescents treated in this series were minimal.

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THE COMBINATION OF ACTH-CORTISONE-HYDROCORTISONE WITH ANTIBIOTICS IN THE MANAGEMENT OF OVERWHELMINGLY SEVERE INFECTIONS

Theory and Practice Based on Three Years' Experience

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IN THE course of the past three years, the administration of ACTH-cortisone in conjunction with antibiotics to patients with very severe medical and surgical infections, apparently inadequately responsive to antibiotics alone, has been subjected to critical evaluation in a group of eighty-three patients in this institution. Several brief reports of this work have already appeared.[1, 2] The present report represents a summary of our total experience with such therapy in this hospital during the period 1950 to 1953. With few exceptions, the patients who have received such combined hormonal-antibiotic therapy have been individuals who, in the opinion of the attending physicians, were in such poor physical condition that their survival seemed improbable with "standard" therapeutic regimens. This judgment was not necessarily correct in all instances, i.e., recovery of the patient on combined therapy does not necessarily indicate that recovery might not have occurred on antibiotic therapy alone.

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THERAPEUTIC PROGRAM

As noted below, hormonal therapy has been administered to this type of patient to permit of his survival until such time as antibiotic therapy has time to take effect. For this reason, *intensive* hormonal therapy has been used in essentially all instances.

In Fig. 1 are shown the two dosage schedules which are currently being followed in such patients. Arbitrarily, patients who are "quite ill" are placed on the short-term schedule; those who are "extremely ill" receive hormonal therapy for seven days. Such a division obviously is by no means absolute, and depends upon the observer's judgment. In all pa-

tients, one discontinues hormone administration as soon as possible in order to avoid the danger of dissemination of infection which might ensue if hormonal therapy were continued for too long a period. The choice of antibiotics rests upon clinical and laboratory findings, including in vitro sensitivity tests when available. *Antibiotic therapy is always continued for at least three days following the discontinuance of hormone administration!*

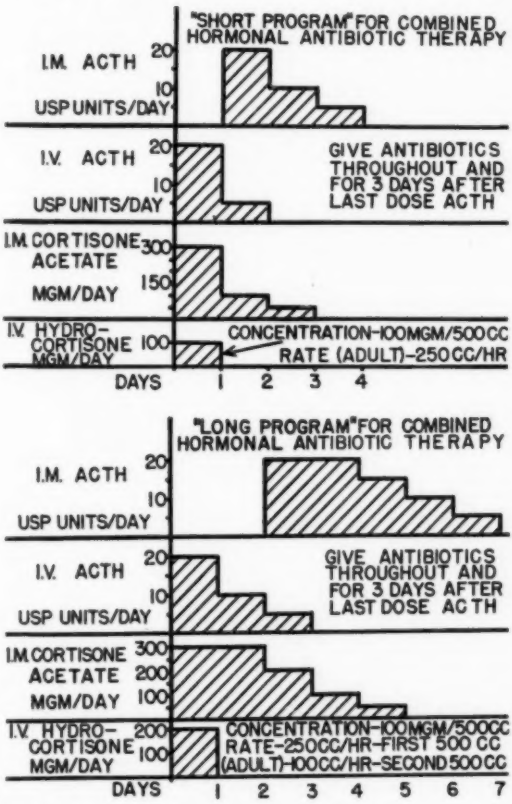

Fig. 1.—Short- and long-term programs for combined hormone-antibiotic therapy.

CLINICAL OBSERVATIONS

In Table I are presented the summaries of forty-two medical cases treated in the manner described above. Of the forty-two, twelve received immediate, and obvious, benefit; six appeared to be significantly benefited; the results were uncertain in seventeen; in seven, there was probably no benefit. Twelve of the patients died. In one of the two cases of meningococcemia associated with Waterhouse-Friderichsen syndrome, cortisone but no ACTH was administered. There was a good initial response. Diarrhea, circulatory collapse, and death ensued in rapid succession on the seventh day following withdrawal of cortisone therapy. Post-mortem examination revealed

TABLE I. MEDICAL CASES.

NAME	AGE	CLINICAL DIAGNOSIS AND FINDINGS	DURATION PRE-HORMONES	SEVERITY PRE-HORMONES	DURATION HORMONE ADMINISTRATION	RESPONSE TO HORMONES: TEMPERATURE FALL	RESPONSE TO HORMONES: CLINICAL	FINAL STATUS	MORBIDITY	BENEFICIAL ROLE OF HORMONES
				Meningitis						
AND	65 yr.	Meningococcal	3 da.	Drowsy Hypotension	3 da.	Prompt	2+	3+	Mental confusion Slow convalescence	Unknown
CUR	23 mo.	Meningococcal	8 da.	Moderate illness, eye impairment	8 da.	Prompt	3+	2+	Vitreous hemorrhage Joint pain and swelling	Definite
GAR	2 yr.	Meningococcal with petechiae	2 da.	Lethargic	9 da.	Prompt	3+	4+	Herpetic stomatitis post-hormone	Unknown
HAY	20 yr.	Meningococcal with petechiae	3 da.	Stuporous Moderate hypotension	5 da.	Prompt	4+	4+	—	Probable
HEN	14 yr.	Meningococcal	2 da.	Stuporous shock	20 da.	Prompt	3+	3+	Recurrent hypotension Muscle weakness	Definite
MEN	39 yr.	Meningococcal	7 da.	Critical	6 da.	Normal 48 hr.	2+	3+	Moderate edema	Unknown
MURC	21 yr.	Meningococcal with petechiae	2 da.	Comatose	6 da.	Slow	3+	4+	Sterile effusion of elbow	Unknown
ORT	3 yr.	Meningococcal	4 da.	Critical	8 da.	Slow	±	3+	Joint swelling; hematuria	Unknown
POR	10 mo.	Meningococcal with petechiae	36 hr.	Lethargic	6 da.	Normal 48 hr.	4+	4+	—	Unknown
STE	48 yr.	Meningococcal with petechiae	2 da.	Comatose	3 da.	Prompt	2+	2+	Purulent arthritis of knee; facial weakness, left	Unknown
WAT	6 yr.	Meningococcal with petechiae	2 da.	Lethargic toxic	4 da.	Prompt	4+	4+	—	Probable
LOB	14 mo.	Meningococcal with petechiae	36 hr.	Comatose Convulsing	2.5 da.	0	0	Expir.	Pulmonary edema Bronchopneumonia	None
DAB	22 mo.	*H. influenza*	18 hr.	Stuporous Convulsing	6 da.	Slow	3+	4+	Muscle flaccidity; gradual return to normal	Definite
HARD	20 mo.	*H. influenza*	5 da.	Critical	20 da.	Prompt	3+	4+	—	Definite

NAT	1 yr.	*H. influenza*	18 da.	Comatose	9 da.	Slow	2+	4+	Initially blind; later full recovery	Unknown
KEM	28 yr.	Brain abscess and meningitis	14+ da.	Progressive deterioration	7 da.	Prompt	4+	4+	—	Definite
MIL	14 yr.	Etiology unknown purulent C.S.F.	1 da.	Comatose Hyperthermia	6 da.	Prompt	0	Expir.	Respiratory arrest post-24 hr., encephalomalacia	None
MURP	68 yr.	Purulent C.S.F. Pneumonia	5 da.	Comatose	3 da.	Slow	0	Expir.	Bronchopneumonia ophthalmitis	None
				Meningococcemia Without Meningitis						
BEN	8 mo.	Necrotic purpuric skin lesions	1 da.	Comatose Convulsing Shock	4 da.	Normal 36 hr.	3+	Expir.	Diarrhea—adrenal failure 7 da. post-treatment	Probable (initially)
DEG	13 mo.	Petechiae	10 hr.	Stuporous shock	10 hr.	0	0	Expir.	Probable adrenal hemorrhage and failure	None
				Pneumonia						
BAC	3 yr.	Bronchopneumonia (diffuse)	4 da.	Lethargic Toxic	5 da.	Prompt	4+	4+	Congestive heart failure (moderate)	Definite
NOR	54 yr.	Bronchopneumonia	3 da.	Comatose Cyanotic	4.5 da.	Prompt	3+	4+	—	Definite
POW	59 yr.	Bronchopneumonia (steptococcal)	1-3 wk.	Critical	11 da.	Normal 3 da.	2+	4+	—	Probable
SHE	46 yr.	Lobar (pneumococcal)	4 da.	Stuporous Moribund	5 da.	Prompt	4+	3+	Pulmonary abscess; subsequent lobectomy	Definite
YOU	68 yr.	Bronchopneumonia	7 da.	Moribund	12 hr.	None	0	Expir.	—	None
				Miscellaneous Infectious Diseases—Bacterial Etiology						
LEA	35 yr.	Acute endocarditis (Hemolytic *Staph. aureus*)	22 da.	Very toxic poor response to antibiotics	9 da.	Prompt	±	Expir.	Myocarditis; perforated mitral valve; brain abscess; renal abscesses; pneumonia	Unknown
OLI	47 yr.	Tetanus; marked muscle spasm	9 da.	Critical	3 da.	Slow	2+	Expir.	Laryngospasm; focal hyalinization of adrenals	Probable (initially)

TABLE I—CONT'D

NAME	AGE	CLINICAL DIAGNOSIS AND FINDINGS	DURATION PRE-HORMONES	SEVERITY PRE-HORMONES	DURATION HORMONE ADMINISTRATION	RESPONSE TO HORMONES		FINAL STATUS	MORBIDITY	BENEFICIAL ROLE OF HORMONES
						TEMPERATURE FALL	CLINICAL			
BLA	68 yr.	Tetanus; severe muscle irritability trismus?	8 da.	Critical	10 hr.	None	0	Slow recovery	Bronchopneumonia	None
DUR	39 yr.	Diphtheria; myocarditis	14 da.	Critical	5 da.	None	1+	Expir.	Edema; atelectasis	Unknown
EAS	50 yr.	Diphtheria; myocarditis	10 da.	Critical	5 da.	None	±	Expir.	Pulmonary edema	Unknown
HEW	46 yr.	Botulism	4 da.	Progressive bulbospinal involvement	11 da.	No fever	4+	4+	Tracheotomy and respirator at onset of treatment	Unknown
		Miscellaneous Infectious Diseases—Viral Etiology								
DON	19 yr.	Infectious mononucleosis with hepatitis	20 da.	Unresolving hepatitis; moderately ill	5 da.	Afebrile	4+	4+	—	Probable
JUN	52 yr.	Homologous serum hepatitis	20 da.	Rapid deterioration; toxic		Afebrile	4+	4+	—	Definite
LOV	46 yr.	Homologous serum hepatitis	24 da.	Rapid deterioration; comatose	11 da.	Prompt	4+	4+	—	Definite
GRI	4 yr.	Influenza; (initial diagnosis meningitis)	3 da.	Hyperthermia convulsing	4 da.	Prompt	4+	4+	—	Unknown
KEE	21 mo.	Bronchiolitis; bronchopneumonia	3 da.	Marked fatigue	3 da.	Prompt	4+	4+	—	Unknown
WAR	1 yr.	Bronchiolitis	4 da.	Critical Toxic	10 da.	Prompt	2+	Expir.	Septicemia	Definite (initially)
BRO	3 yr.	Bulbospinal poliomyelitis	10 da.	Critical	7 da.	Slow	3+	4+	—	Unknown
HARP	21 yr.	Poliomyelitis; resp. paralysis	14 da.	Moribund	5 da.	None	0	Expir.	—	None
WES	18 yr.	Poliomyelitis; transverse myelitis	7 da.	Toxic hyperthermia	2 da.	Prompt	2+	2+	Complete paraplegia marked sensory loss	Unknown
SAA	24 yr.	Measles; encephalomyelitis; paraplegia	8 da.	Critical	6 da.	Afebrile	±	4+	Decubitus	Unknown
WILLS	34 yr.	Mumps; orchitis; pancreatitis; encephalitis	8 da.	Acute stage orchitis	5 da.	Prompt	4+	4+	—	Definite

atrophic adrenal cortices. Such an occurrence probably would have been prevented by the present "standard" regimen, where ACTH is continued for at least twenty-four hours following the discontinuance of cortisone.

Table II presents data on forty-one surgical cases. All but one of these cases represent peritonitis associated with primary abdominal or pelvic pathology. In this group there were patients with gastric perforations and anastomosis breakdown have been less satisfactory. In the former group, the addition of hormonal therapy has permitted early surgery, and has usually been associated with an uncomplicated postoperative convalescence.

In Figs. 2 and 3 are shown rather typical clinical courses and temperature curves in medical and surgical

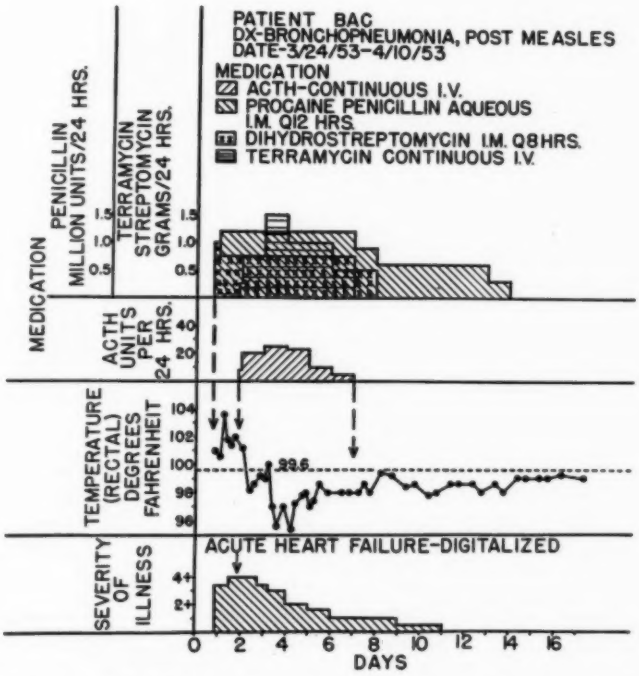

Fig. 2.—Clinical course of a 3-year-old boy with post-measles bronchopneumonia, showing response to hormonal therapy.

sixteen "definitely beneficial effects" of hormonal therapy, twelve probable, two uncertain, and eleven cases with improbable effect. Seventeen patients died. Of the patients included in this series, those with ruptured appendices have shown the most striking response, while the final result in patients, respectively. The first case is that of a 3-year-old boy who entered the hospital with a temperature of 102.6° F., persistent lethargy, and a nonproductive cough ten days following the onset of measles. Respirations and pulse rate were rapid and both lungs revealed diffuse râles and

TABLE II. SURGICAL CASES

NAME	AGE	CLINICAL DIAGNOSIS AND FINDINGS	DURATION PRE-HORMONES	SEVERITY PRE-HORMONES	DURATION HORMONE ADMINISTRATION	RESPONSE TO HORMONES: TEMPERATURE FALL	RESPONSE TO HORMONES: CLINICAL	FINAL STATUS	MORBIDITY	BENEFICIAL ROLE OF HORMONES
Ruptured Appendix										
BER	2½ yr.	Generalized peritonitis Bronchopneumonia	3 da.	Toxic and moribund	(a) 12 da. (b) 6 da.	(a) Normal 48 hr. (b) Prompt	3+ 4+	4+	Pelvic abscess—rupture with 2nd episode generalized peritonitis	Definite
BES	73 yr.	Appendiceal abscess; small bowel abscess; perforation terminal ileum	22 da.	Debility and shock	3 da.	Prompt	4+	4+	Wound abscess	Definite
CAR	5 yr.	Diffuse suppurative peritonitis	2 da. 2 da.	Lethargic Toxic	5 da.	Prompt	4+	4+	Small stitch abscess	Probable
ELY	7 yr.	Generalized peritonitis	Surgery 2 da. after hormone	Critical Toxic	8 da.	Prompt	4+	4+	None	Definite
MCN	4 yr.	Generalized peritonitis	24 hr.	Lethargic Toxic	4 da.	Normal 48 hr.	3+	4+	Febrile after D.C. hormone	Definite
PER	42 yr.	Diffuse suppurative peritonitis	24 hr.	Critical	4.5 da.	Prompt	4+	4+	—	Unknown
SMI	8 yr.	Diffuse suppurative peritonitis	20 hr.	Critical	6 da.	Normal 48 hr.	4+	4+	Superficial wound sepsis	Unknown
SPA	2½ yr.	Adherent RLQ abscess	5 da.	Critical Toxic	5 da.	Normal 36 hr.	4+	4+	Surgery postponed until 14 da. post-D.C. ACTH-Ochsner regimen	Definite
ARA	8 yr.	Adherent reaction RLQ; ruptured appendiceal abscess; ileus	24 hr.	Acute distress	7 da.	Prompt	4+	4+	—	Definite

Gastroduodenal Perforation										
GIL	36 yr.	Conc. H_2SO_4 ingestion; massive gastric necrosis; diffuse chemical burns	6 hr.	Shock	6 da.	Good	3+	Expir.	Necrosis duodenum with perforation	Definite (initially)
LOC	54 yr.	Pyloric ulcer; generalized peritonitis	15 hr.	ō blood pressure; moribund	11 da.	ō	ō	Expir.	Perforated gastric ulcer adjacent to closure	None
WILLI		Jejunal ulcer; generalized peritonitis	24 hr.	Shock	9 da.	Fair	2+	Expir.	Left subphrenic abscess drained; fistula formation	None
MAK	68 yr.	Carcinoma-stomach; generalized peritonitis	?	Shock	3 da.	Afebrile	4+	Expir.	Refused surgery	Probable (During treatment)
CAL	67 yr.	Gastric ulcer; perigastric abscess	5 da.	Shock Cyanosis	2 da.	ō	ō	Expir.	Old subphrenic abscess; recent perigastric abscess	None
HIG	70 yr.	Duodenal; generalized peritonitis	16 hr.	Disoriented Critical	4 da.	Good	4+	4+	—	Probable
LYL	39 yr.	Prepyloric; generalized peritonitis	20 hr.	Critical	4 da.	Prompt	4+	4+	—	Definite
PER	52 yr.	Pyloric; generalized peritonitis	14 hr.	Critical	3 da.	Prompt	3+	4+	Perforated gastric ulcer on 14th postoperative day	Probable
Small Bowel Perforation										
OLL	40 yr.	Strangulated small bowel; generalized peritonitis	18 hr.	Critical	4 da.	Prompt	4+	4+	—	Definite
SHA	64 yr.	Small bowel obstruction; localized peritonitis	15 hr.	Critical	5 da.	Prompt	4+	4+	—	Definite
SLA	47 yr.	Volvulus; generalized peritonitis	?	Toxic Shock	3 da.	Good	4+	3+	Mental depression; parotitis; pneumonia; pulmonary infarction	Probable

TABLE II.—CONT'D

NAME	AGE	CLINICAL DIAGNOSIS AND FINDINGS	DURATION PRE-HORMONES	SEVERITY PRE-HORMONES	DURATION HORMONE ADMINISTRATION	RESPONSE TO HORMONES		FINAL STATUS	MORBIDITY	BENEFICIAL ROLE OF HORMONES
						TEMPERATURE FALL	CLINICAL			
Ruptured Cecum										
BEA	65 yr.	Carcinoma of colon; peritonitis and gross fecal contamination	2 da.	Toxic Shock	7 da.	Prompt	4+	Expir.	Severe electrolyte imbalance; autopsy: purulent pneumonia	Definite (initially)
BOE	44 yr.	Fecal impaction; generalized peritonitis; fecal contamination	24 hr.	Critical	4 da.	Normal 36 hr.	4+	4+	—	Definite
MAT	87 yr.	Carcinoma of sigmoid colon; generalized peritonitis	24 hr.	Toxic Stuporous	8 da.	Good	3+	Expir.	Necrosis of cecum and rupture	Probable (initially)
MEY	56 yr.	Plastic peritonitis; probable ruptured cecum	16 hr.	Toxic Shock	4 da.	Brief	2+	Expir.	Gangrenous proximal ascending colon	None
Ruptured Colon										
BAR	26 yr.	Constricting colon lesion; generalized peritonitis	24 hr.	Hypotensive	6 da.	Normal 36 hr.	4+	4+	Cul-de-sac abscess	Definite
CAP	40 yr.	Lymphopathia with stricture of rectum; localized peritonitis	?	Shock Toxic	10 da.	Prompt	3+	3+	Debility; slow convalescence	Probable
OWE	73 yr.	Neurolemmosarcoma generalized peritonitis	10 da.	Toxic; hyperthermia	5 da.	Prompt	3+	Expir.	Debility	Probable (initially)
SED	64 yr.	Perforated sigmoid-post-detortion of volvulus; localized peritonitis	18 hr.	Moribund Shock	6 da.	Prompt (initially)	±	Expir.	Bronchopneumonia	None
Breakdown Following Bowel Resection										
AML	86 yr.	Ileum resection; generalized peritonitis	24 hr.	Critical	7 da.	Prompt	3+	Expir.	Rebreakdown	Probable (initially)

BAR	2 yr.	Sigmoid colon resection; localized peritonitis	?	Toxic, no response to antibiotics	(a) 7 da. (b) 5 da.	Prompt	4+	4+	Abdominal dehiscence during first course of treatment	Definite
OGG	57 yr.	Colon pouch mobilization; duodenal blowout; generalized peritonitis	36 hr.	Shock Moribund	3 da.	Prompt	±	Expir.	Pericarditis; atelectasis	None
TAY	64 yr.	Carcinoma of colon; colostomy; localized peritonitis	24 hr.	Debilitated Toxic	(a) 6 da. (b) 5 da.	Prompt	(a) + (b) ō	Expir.	Dehiscence and perforation of carcinoma	None
TOR	73 yr.	Resection of carcinoma of colon; generalized peritonitis	24 hr.	Shock	2.5 da.	Afebrile	±	Expir.	Low salt syndrome; renal shut-down; unable to explore	None
WAS	76 yr.	Resection of carcinoma of colon; generalized peritonitis	24 hr.	Shock	1 da.	ō	ō	Expir.	No response; unable to explore	None
					Ruptured Gall Bladder and Bile Peritonitis					
BUR	75 yr.	Bile peritonitis post-liver biopsy	2 da.	Shock Toxic	2 da.	2+	2+	Expir.	—	None
CAL	77 yr.	Acute cholecystitis generalized peritonitis	24 hr.	Shock Toxic	10 da.	Prompt	3+	3+	—	Probable
GRA	75 yr.	Acute cholecystitis generalized peritonitis	24 hr.	Critical	3 da.	Prompt	4+	4+	—	Probable
STE	74 yr.	Cholecystitis; localized peritonitis	5 da.	Toxic	4 da.	ō	ō	Expir.	RUQ retroperitoneal abscess	None
					Ruptured Pelvic Abscess					
MCF	60 yr.	Perivesicular abscess; localized peritonitis	12 da.	Toxic Downhill course	5 da.	Good	3+	4+	—	Definite
PHI	31 yr.	Bilateral pyosalpinx; regional peritonitis	4 da.	Unresponsive	2 da.	Good	4+	4+	—	Definite
					Miscellaneous Nonperitonitis					
FIL	9 yr.	Palmar abscess septicemia	4 da.	Severe toxic reaction	6 da.	Prompt	4+	4+	Repeat I and D simultaneous with hormone treatment	Probable

wheezes. A chest roentgenogram revealed increased peribronchial markings bilaterally, as well as widespread fine patchy densities. Combined penicillin and streptomycin therapy was started on entry. Because of progressive deterioration, hormonal therapy uneventfully and hormonal therapy and digitalis were discontinued four days later. No complications were encountered during the remainder of antibiotic therapy, and chest roentgenograms revealed rapid clearing of the diffuse inflammatory process.

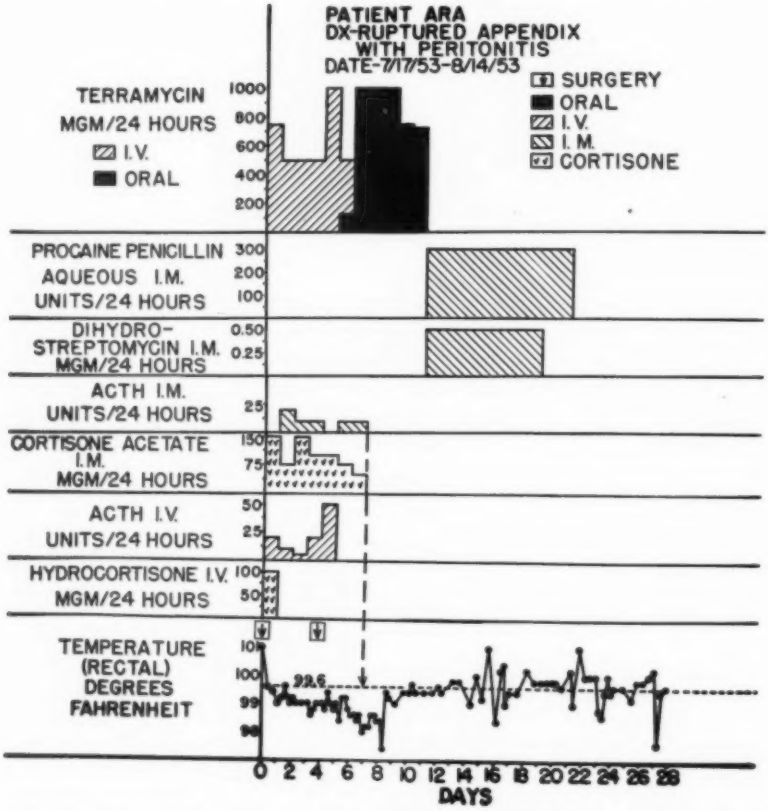

Fig. 3.—Clinical course of a 7-year-old boy with severe generalized peritonitis secondary to a ruptured appendix. Hormonal-antibiotic therapy permitted "early" definitive surgery, and was associated with an uncomplicated convalescence.

plus intravenous Terramycin was instituted twenty-four hours later, together with intravenous digitalization. In the ensuing twenty-four hours there was a dramatic fall in temperature and major clinical improvement. Convalescence continued

The second case is that of a 7-year-old boy. On admission he was extremely toxic with a rigid silent abdomen and a high fever. The history suggested a ruptured appendix two months previously without any therapy at that time, and a second peri-

toneal insult twenty-four hours before admission. Surgery was limited to the insertion of a drain because of the great amount of inflammation, edema, and friability. Intensive hormonal-antibiotic therapy was administered, and three and one-half days later the abdomen was reopened. A large mass was found to be adherent to the jejunum and cecum, and the jejunum was partially obstructed. An appendectomy was performed after extensive blunt dissection, and large raw surfaces remained on the involved intestine. All pus was evacuated and the abdomen was closed without drains. The postoperative course was very comparable to that expected of a patient with a simple, uncomplicated appendectomy. It will be noted that there was moderate temperature elevation following discontinuance of hormonal therapy. This was not associated with any change in the clinical picture, and eventually subsided.

In Fig. 4 is shown the clinical course of a 2½-year-old girl, which illustrates some of the complications which may arise if a precise schedule is not adhered to. The history and findings suggested a probable onset of acute appendicitis four days prior to entry with rupture and general peritonitis appearing two days later. On admission she was extremely toxic, and became moribund during the first twenty-four hours of antibiotic therapy. Surgery was deemed impossible, and hormonal therapy was begun. During the next twenty-four hours, the child became more responsive, the blood pressure stabilized, the bowel sounds became active, and abdominal rigidity decreased to a minimum. The presence of bronchopneumonia prevented surgical intervention until the seventh hospital day. At the time of surgery, the peritoneal cavity was found to contain much purulent fluid, although peritoneal inflammation was negligible except at

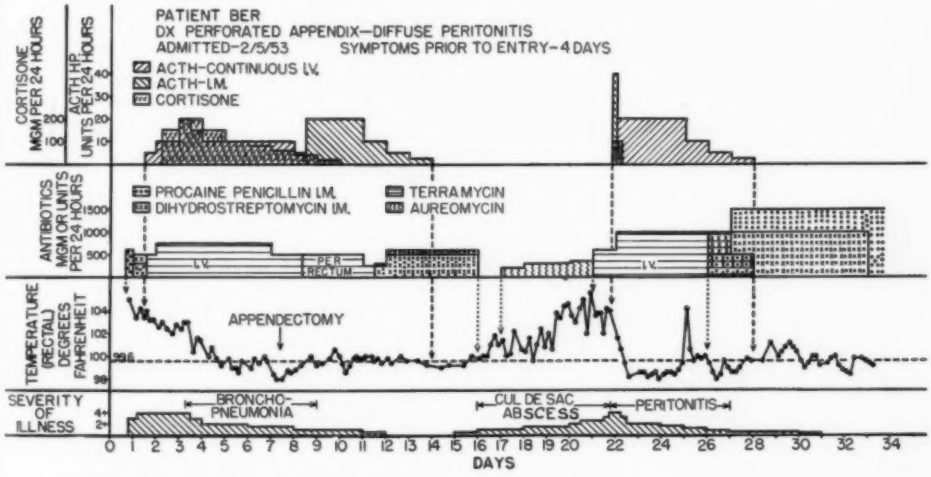

Fig. 4.—Clinical course of a 2½-year-old girl with ruptured appendix and generalized peritonitis, showing good initial response but subsequent complications resulting from inadequate postoperative antibiotic therapy.

the site of the appendiceal rupture. An appendectomy was performed and the abdomen closed without drainage. The postoperative course was benign, and hormonal therapy was stopped on the seventh postoperative day. Antibiotics were inadvertently discontinued two days later and then restarted at inadequate levels after a hiatus of thirty-six hours. During the next four days, the patient became increasingly ill, and showed localizing signs suggesting a beginning cul-de-sac abscess. With re-exploration in mind, hormonal therapy was reinstituted. Response was almost immediate, and surgery was deferred as the signs of generalized peritonitis subsided. Following withdrawal of hormonal therapy, intensive antibiotic treatment was continued for ten days and the patient was finally discharged on the fiftieth hospital day. *It should be emphasized that the decision to continue hormonal therapy without definitive surgical treatment was hazardous,* in that progressing infection might well have been masked by such a regimen.

In Fig. 5 is shown the rapid decrease in jaundice (associated with marked clinical improvement) in a patient with severe homologous serum hepatitis, who prior to the administration of ACTH had shown a progressively downhill course. This represents the usual pattern of response to hormonal therapy in patients with unusually severe viral hepatitis. As one might expect, the response is less predictable in patients with hepatic insufficiency resulting from chemical toxins or associated with chronic alcoholism.

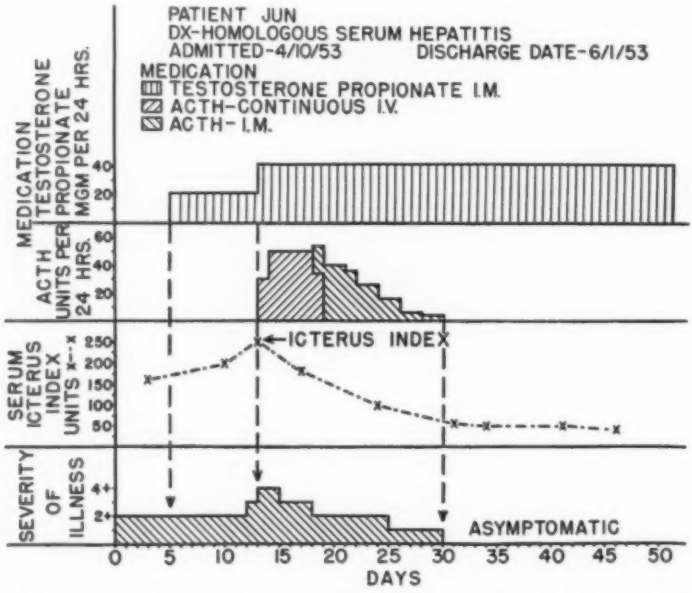

Fig. 5.—Relationship of hormonal therapy to clinical course and icterus index in an adult male with severe homologous serum hepatitis.

DISCUSSION

Long before the availability of ACTH or cortisone for clinical evaluation, a number of clinicians and investigators had been aware that the adrenal cortex played a role of some real importance in processes of immunity and resistance to disease. It was well known that the patient with Addison's disease was extremely sensitive to even minor infections, and further that the administration of whole adrenocortical extract was of value in controlling infection in such patients. Dougherty and White[3] demonstrated that adrenal extracts produced a fall in the monocytic cells of the blood, and presented evidence which they believed was compatible with the concept that cortical hormones produced a lysis of such cells, with consequent release of specific immune globulins.

Kass and associates[4] demonstrated the prompt defervescence and disappearance of toxicity in patients with pneumococcus pneumonia who received ACTH, despite the continuance of growth of organisms in the blood stream in such patients. Smadel and co-workers[5] and Woodward and associate[6] showed that the administration of cortisone to patients with typhoid fever produced a similar disappearance of fever and toxicity.

Many of the animal observations, however, pointed in a different direction. Mogabgab and Thomas[7] demonstrated that the administration of cortisone or ACTH to experimental animals, injected intracutaneously with streptococcus type A organisms of low pathogenicity, increased the mortality rate in such animals. These observations have been confirmed and amplified by many investigators with both bacterial and viral organisms. Of these, the reports of Glaser,[8] Antopol,[9] and Shwartzman,[10] and their respective co-workers are of particular interest. Confronted with the above confusing and apparently conflicting observations, the question obviously arises: What are the mechanisms of these effects?

Sufficient information is at hand to provide a partial explanation:

1. *ACTH and cortisone, in all species studied, have a profound anti-inflammatory effect.* This was first shown by Hench and associates[11] in their original observations on the effect of cortisone and ACTH on rheumatoid arthritis. The suppression of inflammation was characterized by a lessening in the clinical signs; histologically by a decreased number of plasma cells and lymphocytes, a reduction of papillary tufting, a decreased deposition of fibrin, and diminution of necrosis and edema of the synovial membrane. Their observations have been confirmed and augmented by many investigators, who have demonstrated suppression of the inflammatory response to such agents as chemical irritants, foreign proteins and microorganisms. Using skin-window techniques in the human subject, and ear-window techniques in rabbits, Rebuck and collaborators[12] and Ebert and Barclay[13] have demonstrated modification or suppression of the inflammatory reaction by appropriate hormone administration. The precise mechanism of this anti-inflammatory effect of cortical steroids is unknown, but it would appear to be attributable to a direct effect of the hormone upon the involved tissues, with a resulting de-

crease in vascular permeability, diminished exudation, and lessened migration of inflammatory cells. This concept is well borne out in the human subject by the relief obtained in inflamed joints following the injection of hydrocortisone, or by the rapid improvement in an inflamed eye following the local application of cortisone or hydrocortisone.

2. *In a clinical sense, the anti-inflammatory effect may be good or bad.* In experimental animals, the increased susceptibility to a variety of organisms is largely or entirely attributable to this effect, i.e., dissemination of bacteria occurs as the result of lack of construction of "inflammatory walls." This can be equally true in the human being in such diseases as tuberculosis, in which the inflammatory component represents a major part of the body resistance to the infectious process.[14]

The desirable effects are easily demonstrated in such diseases as iritis or mumps orchitis, in which the unchecked inflammatory component can result in complete destruction of an important organ. In our opinion and experience, the same statement can be made in such diseases as severe meningitis or peritonitis. In the former case, inhibition of inflammation may prevent irreversible damage to the central nervous system. *Coadministration of an adequate antibiotic prevents dangerous dissemination, and results in eventual destruction of the infectious agent.* In peritonitis, control of the inflammatory process permits of adequate and early surgery, and prevents major and permanent structural changes in the abdominal viscera.

3. *Cortical steroids per se exert neither inhibitory nor stimulating effects upon the growth of pathogenic microorganisms.* Experimental studies reported by Glaser and associates,[8] and Germuth and collaborators[15] demonstrate this quite well. However, as noted above, the alteration of the inflammatory response by cortical steroids does permit a greater proliferation and dissemination of bacteria in the absence of antibacterial drugs.

4. *The relationship of the hormones to the production of specific antibodies is controversial.* Kass and co-workers[4] found that the time of appearance of antibodies and their titer was of the same degree in patients with pneumococcal pneumonia receiving ACTH as in patients who recover spontaneously from similar infections. Under some experimental conditions, antibody production may be diminished. This has been demonstrated by quantitative immunochemical means in rabbits using polyvalent pneumococcal antigen by Bjørneboe and associates.[16] Fischel and collaborators[17] have found this effect to be more striking on the expected high rate of antibody synthesis in the secondary response (specific amnestic) where rabbits were re-exposed to previously injected egg albumin. The observations of Germuth and colleagues[18] that ACTH and cortisone suppress the active Arthus phenomenon in rabbits further support their findings. On the other hand, cortisone did not inhibit a local passive Arthus nor increase the rate of antibody disappearance. Thus the reduced antibody levels occurring in active Arthus were the result of suppression of antibody formation rather than a more rapid breakdown of antibody protein.

This latter finding was also demonstrated by Fischel and his group.[19] Mirick[20] has shown that cortisone and ACTH fail to suppress antibody formation following vaccination of human subjects with a mixture of pneumococcal polysaccharide. Species differences and variation in hormonal dosage may explain some of the apparent contradictions. *There is at present no evidence that in the well-nourished human being with a severe infection, who is receiving antibiotics, hormonal therapy in any way interferes with specific antibody formation.*

In publications from this laboratory, the term "nonspecific antitoxic effects of ACTH and adrenal steroids" has been used to describe the changes observed in human subjects so treated.[1a] In a clinical sense, the term is used advisedly. How correct it may be in a biochemical or an immunological sense is still to be demonstrated. There appears to be general agreement that these hormonal agents exert little or no protective effect against the exotoxins of diphtheria and tetanus. There is, however, good evidence that a protective effect is exerted against certain endotoxins of bacterial origin.[21a, b] It is still uncertain how much of the "clinical antitoxic effect" of ACTH and cortisone is referable to modification of the inflammatory (and allergic) reaction (with consequent diminution of absorption of the products of inflammation), and how much is referable to literal nonspecific neutralization of specific toxins. It seems probable that this problem will be clarified in the near future.

Whatever the final answers to the above may be, we believe that our observations and the observations of others during the past three years support the following statements:

1. There is no question but that the administration of ACTH or cortisone in sufficient amounts to patients with severe infections resulting from widely diverse organisms will result in rapid and striking clinical improvement (i.e., lessened systemic toxicity).

2. Despite the apparent clinical improvement resulting from such treatment, the organisms causing the disease continue to flourish, and unless eliminated either by specific immune substances and/or by specific antibiotics, will eventually result in the destruction of the patient.

3. For these reasons, ACTH and cortisone (always in conjunction with suitable antibiotics) should be administered to patients with infections only under the following conditions:

a. In nonsurgical diseases where there is excellent reason to believe that the infection will not respond adequately to antibiotics alone.

b. In surgical conditions only when definitive therapy (surgery) is scheduled for the immediate future.

c. Hormone administration in the presence of infection should always be administered for the shortest possible time compatible with adequate clinical response, and must always be discontinued *before* discontinuance of antibiotics. Also, hormone administration must always be accompanied with adequate adjuvant measures (diet, potassium, testosterone, estrogen, etc.)[22]

4. As in the case of all rules in clinical medicine, certain exceptions must be made to statement 3. In our

experience thus far, we believe that there are at least three proper exceptions:

a. All patients with meningococcal meningitis should receive hormonal therapy. With the exception of the one child in whom cortisone alone (rather than ACTH plus cortisone) was administered, and in whom adrenal atrophy and death ensued, the good results have been most impressive, and if untoward effects have occurred, we have failed to recognize them.

b. No antibiotic is available for the virus of hepatitis. Yet it would appear that the administration of ACTH and cortisone to patients with severe forms of this disease may be lifesaving, and will beyond question produce a striking clinical improvement. It also abets the objective of optimal food intake, which, in the patient with liver damage, is of profound importance.

c. In patients with mumps orchitis, again no specific antibiotic is available. Yet on the basis of limited experience of one of us (L. W. K.), it appears that adequate hormonal therapy will preserve the integrity of the testicle and will produce no untoward effect.

SUMMARY

Data are presented which may support the concept that the addition of ACTH and cortisone therapy to well-indicated antibiotic therapy in patients suffering from severe infections results in reduction of morbidity and mortality in such patients. Such combined therapy should be used with a full knowedge of the potential hazards as well as the potential assets involved.

Grateful acknowledgment is made to The Armour Laboratories for supplies of ACTH, to Merck & Co., Inc., for supplies of cortisone and hydrocortisone, and to Chas. Pfizer Co. for supplies of Terramycin used in these studies.

The cooperation of the following members of the visiting staff (Department of Surgery) is gratefully acknowledged: Drs. Philip N. Baxter, Arthur J. Hunnicutt, George C. Henegar, and Theodore C. Lawson.

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VARIATIONS IN AGE DISTRIBUTION OF POLIOMYELITIS. COMPARATIVE RATIOS OF YOUNGER AND OLDER AGE GROUPS

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DURING the 1949 epidemic of poliomyelitis in New York City, variations in the age distribution of reported cases were noticed which seemed sufficiently important to warrant further investigation. A review of the literature revealed some evidence that the percentage of cases in the younger age groups showed a tendency to decrease and the percentage in the older groups to increase with the progress of an epidemic.[1-5]

Such data are of epidemiologic interest and might assume practical significance as a source of basic information for evaluating the results of the administration of gamma globulin in epidemic areas where all children under 10 years of age were inoculated. Since no controls were used in such areas in 1953, one method of evaluation would involve a comparison of the trend of the epidemic in children under 10 years of age with those above this age.

In the present investigation 8,964 cases from 1944 to 1952 were studied. Paralytic and nonparalytic cases were included. The minimal diagnostic criteria were at least three of the following findings: fever, stiff neck or back, increased white cell count in the spinal fluid, and flaccid paralysis. The diagnosis was confirmed by a diagnostician who visited every patient about a week after notification.

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Table I gives a summary of the number of cases reported each year.

The case fatality fluctuated from 3.3 per cent in 1952 to 7.3 per cent in 1949, the average for the entire period being 5.6. It was related to age and clinical type of infection. The mortality per 100,000 population varied from 0.1 in 1947 to 2.2 in 1949.

TABLE I

YEAR	NUMBER	RATE PER 100,000
Total	8,964	12.6
1944	1,890	24.6
1945	545	7.1
1946	716	9.2
1947	224	2.9
1948	703	8.7
1949	2,446	31.0
1950	1,064	13.1
1951	555	6.8
1952	821	10.2

The clinical types of infection observed in the two age groups under consideration are shown in Table II. In each age group, about 60 per cent of the cases were paralytic and 40 per cent nonparalytic.

The date of onset was known in 8,851 cases (98.7 per cent) and unknown in 113 cases (1.3 per cent). Analysis by week of onset was based on the known cases. The number reported in the first twenty weeks of the year represented 1.2 per cent of the total. From the twenty-first to the twenty-sixth week, another 2.0 per cent were reported. After the twenty-sixth week, the numbers increased rapidly to a maximum between the thirty-third

and thirty-fifth weeks, when 26.6 per cent of the cases occurred. After that, the number declined slowly, reaching 1.2 per cent of the total in the last four weeks of the year. Since 98.8 per cent of all cases occurred between the twenty-first and fifty-second weeks of the year, the study was confined to this period, i.e., from the latter half of May to the end of the year.

The distribution of cases by week of onset is shown graphically in Chart I. Annual differences are noted in the rate of increase and decline, in the shape of the curves, and in the peak week. The differences became apparent after the twenty-sixth week, and were most marked between the twenty-seventh and fortieth weeks. An early steep rise in cases was associated with a high poliomyelitis season, as in 1949 and 1944. The subsequent decline in the epidemic years was rapid at first, and then slowly tapered off

TABLE II

CLINICAL TYPE	TOTAL NO.	PER CENT	0-9 YR. OF AGE		10 YR. AND OVER	
			NO.	PER CENT	NO.	PER CENT
Total	8,964	100.0	5,309	100.0	3,655	100.0
Paralytic	5,506	61.4	3,283	61.8	2,223	60.8
Nonparalytic	3,458	38.6	2,026	38.2	1,432	39.2

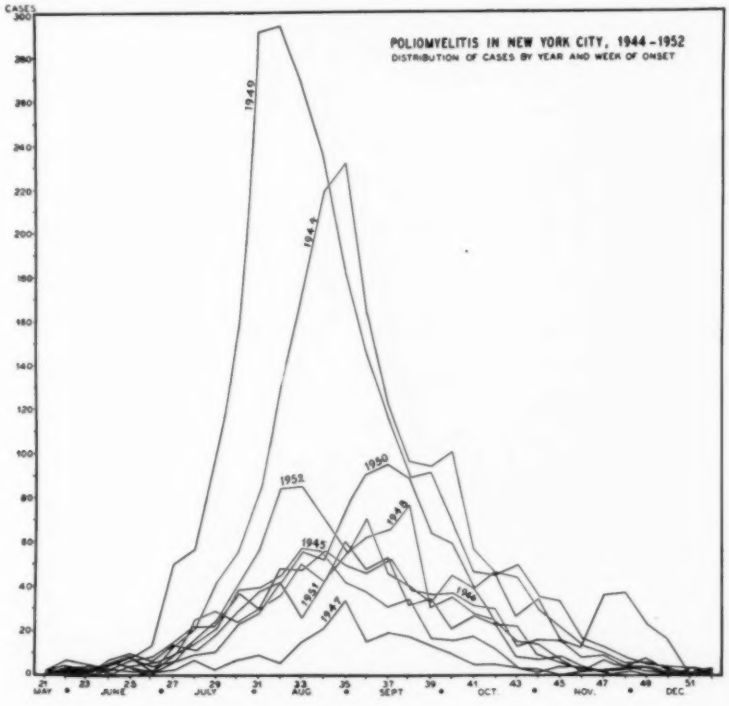

Chart I.

after the thirty-eighth week until it reached endemic levels. The peak for the different years was reached between the thirty-second and thirty-eighth weeks. For all years combined, the peak was in the thirty-fourth week or in the latter part of August.

reached at about the same time by both groups. The close relationship of the two age groups to the annual trend for all ages is reflected in the summary of the data on the peak week for each group by year of occurrence (Table III).

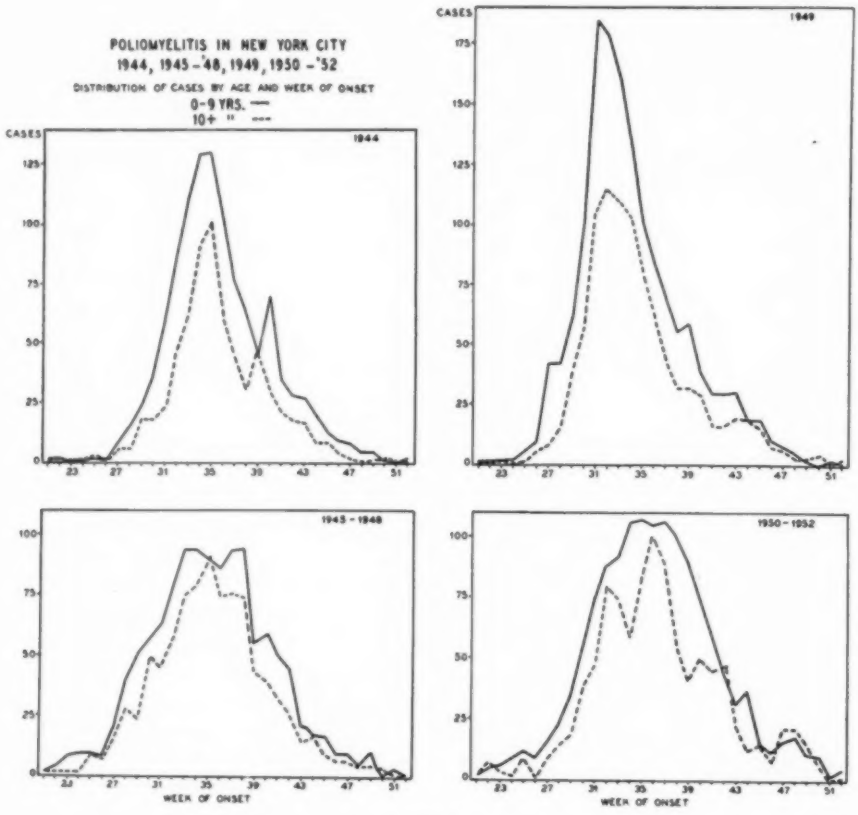

Chart II.

The seasonal fluctuation in cases among those under 10 years of age and those 10 years and over tended to conform to the general pattern of a given year, as shown in Chart II for epidemic and endemic periods. In each of these periods, the crests were

Nevertheless, certain differences in the curves were noted and these seemed worthy of further investigation. This was done by comparing the proportionate distribution of cases by age group during the course of the poliomyelitis season. Since few cases oc-

TABLE III

YEAR	PEAK WEEK BY AGE GROUPS		
	ALL AGES	0-9 YEARS	10 YEARS AND OVER
All years	34	34	35
1944	35	35	35
1945	33	34	33
1946	34	33	35
1947	35	35	35
1948	38	38	38
1949	32	31	32
1950	37	38	37
1951	35	35	35
1952	33	33	32

e.g., 100, 200, or 500 per period; the other, the subdivision of cases into equal time units regardless of numbers reported. The latter method was used because it had the advantage of feasibility, since the cases had been tabulated by week of onset. In addition, one or more time units could be combined when the cases were few or when the effects of certain variable environmental factors were investi-

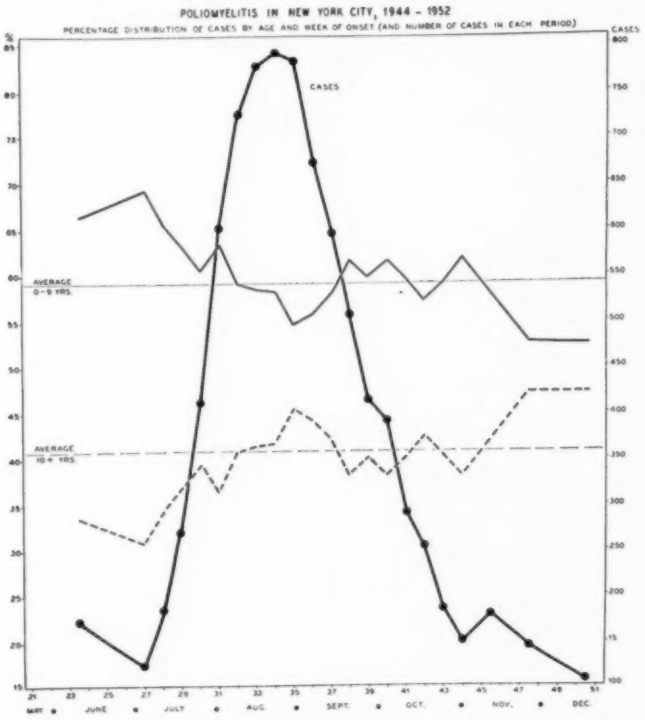

Chart III.

curred at the beginning and end of the season, it became necessary to combine periods or weeks of onset so that the total would not be too small and the sampling error not too large. Two approaches were considered: one, the subdivision of cases into periods encompassing equal numbers of cases, gated, such as school attendance. It had the disadvantage in that the sampling error varied considerably from one time period to another, and the percentage distribution could be misleading if the total number of cases considered was not always kept in mind.

When the entire nine-year period was considered by accumulating corresponding weeks of the year, the number of cases each week exceeded 100 with the exception of weeks 21 to 26 and 45 to 52. It was decided to combine these so that all percentages for the nine-year period would be based on 100 or more cases. This entailed the combination of weeks 21 to 26, inclusive, 45 and 46, 47 and 48, 49 to 52, inclusive.

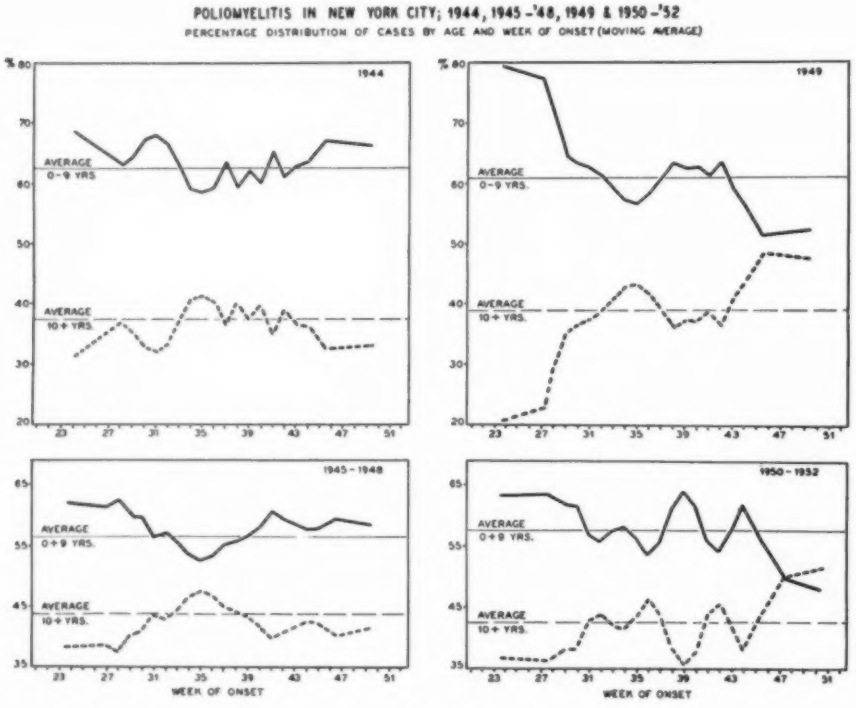

Chart IV.

The percentage age distribution by week of onset from 1944 to 1952, inclusive, is shown in Chart III for those under 10 years of age and those 10 years and over. The former represented 59 per cent of all cases and the latter 41 per cent. For the entire nine-year period, the weekly percentage of cases in the younger group was high early in the poliomyelitis season and then dropped progressively to a low level in midseason. A secondary rise occurred in September, followed by a decline at the end of the year. The reverse trend was observed in the older group.

In studying the results in endemic and epidemic years separately, the minimum number of cases considered in each time unit of one or more weeks was reduced to twenty, and the percentages shown in the charts were based on a three-week or three-period moving average of cases occurring before, during, and after the specific weekly period designated in the chart.

The outcome in the 1944 and 1949 epidemics and in the interepidemic years is shown in Chart IV. The general trend is similar to that noted in Chart III for all years combined. There is a progressive drop in the percentage of cases in children under 10 years of age until about the thirty-fifth week, and a secondary rise thereafter. The reverse is true for the group over 10 years of age.

The percentage distribution of paralytic and nonparalytic cases by age group and week of onset was studied for the years 1949, 1951, and 1952. The data are shown in Chart V. The trends of the curves are generally similar to those previously shown. However, the curve for the nonparalytic cases in 1951-1952 does not indicate a high frequency early in the season for age group 0 to 9 years.

The extent of the difference between younger and older groups in any phase of the poliomyelitis season is measured by the distance between the percentage curves shown in the charts. This naturally fluctuated with time, decreasing during one part of the season and increasing in another.

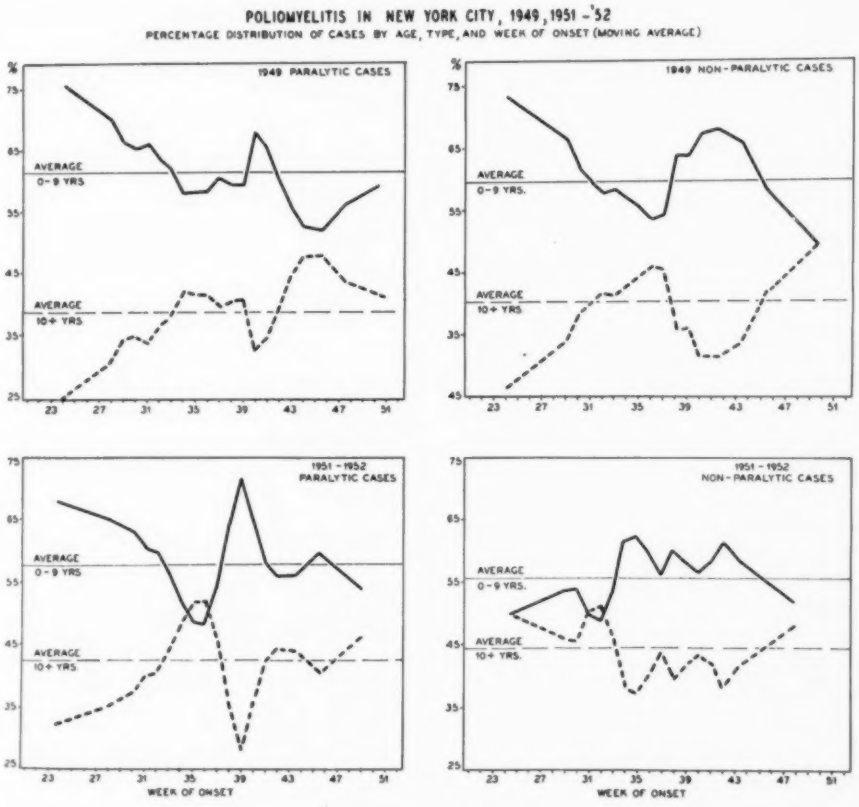
POLIOMYELITIS IN NEW YORK CITY, 1949, 1951 - '52
PERCENTAGE DISTRIBUTION OF CASES BY AGE, TYPE, AND WEEK OF ONSET (MOVING AVERAGE)
1949 PARALYTIC CASES
1949 NON-PARALYTIC CASES
1951 - 1952 PARALYTIC CASES
1951 - 1952 NON-PARALYTIC CASES
AVERAGE 0-9 YRS.
AVERAGE 10+ YRS.
%
WEEK OF ONSET

Chart V.

DISCUSSION

The present report was stimulated by the need of comparative data on the increase or decrease in reported cases of poliomyelitis in age groups 0 to 9 years and 10 years and over as an aid in evaluating the mass prophylactic use of gamma globulin in epidemic areas in 1953. Although annual fluctuations occurred in incidence, time of peak, and epidemic pattern of poliomyelitis in New York City between 1944 and 1952, the curve of newly reported cases was similar for both age groups in any one year and reached a peak in the same week or within one week of the peak for all ages. There appeared, therefore, to be no indication of a difference in time and rapidity of viral invasion of these two broad segments of the host population.

However, a variation in seasonal pattern of the two age groups was demonstrable by an analysis of the percentage distribution of cases by age and week of onset. Although some differences in trends were noted from year to year, there was a fairly consistent decline in the percentage of cases observed in the group under 10 years of age during the first half of the poliomyelitis season and a secondary rise thereafter. The reverse was true for the group over 10 years of age. These changes varied in degree from year to year. They occurred spontaneously in the absence of specific community control measures. Their statistical effect was to alter the extent of the difference between the two age groups from time to time. Therefore, the use of comparative rates of decline in occurrence of cases of poliomyelitis in young age groups after injections of gamma globulin as a test of the efficacy of the injection should be done with caution.

SUMMARY

1. The seasonal fluctuation in cases of poliomyelitis in New York City for those under 10 years of age and those over 10 years was examined from 1944 to 1952, inclusive.

2. Annual variations were observed in the seasonal occurrence of cases. The two age groups examined tended to conform to the pattern of incidence that prevailed in any given year.

3. The trend in the percentage distribution of cases by age and week of onset revealed differences between the two age groups considered. In the younger age group it decreased to a low point in the thirty-fifth week and then increased. In the older age group the reverse trend was noted.

4. The use of a decrease in reported cases of poliomyelitis in young age groups following administration of gamma globulin as a gauge of its effectiveness should be made with caution.

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CAT-SCRATCH DISEASE ASSOCIATED WITH AN OSTEOLYTIC LESION

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IN 1932, Foshay[2] first recognized the occurrence of a regional lymphadenopathy following the scratch of a cat. In 1945, Hanger and Rose[3] produced an antigen from the lesion of a patient with what was thought to be "cat-scratch disease." It was the impetus of this diagnostic tool that led workers in France and the United States to establish what seems to be more than a casual relationship between the scratch of a cat and the presence of a granulomatous regional lymphadenopathy. Debré and associates[1] presented the first report of the subject in 1950. Daniels and MacMurray[2] have presented an adequate review of the entity of cat-scratch disease.

According to Daniels and MacMurray[2] the axillary lymph nodes are the most frequently involved. The cervical lymph nodes are the next most frequently affected group. These authors, however, have emphasized the protean nature of cat-scratch disease. Instances of the disease which have resembled tuberculous adenitis,[3] conjunctivitis associated with pre-auricular lymphadenopathy (oculoglandular disease),[2] and encephalitis[4, 6] have been reported, whereas some patients may suffer from only a mild lymphadenopathy not requiring medical care.

Review of the published reports of cat-scratch disease has failed to reveal a previous instance associated with an osteolytic lesion. Daniels[5] reported the occurrence of a granulomatous osteomyelitis of a digit, directly associated with the bite of a cat, but the patient had a negative skin test after injection of a supposedly potent antigen. The present paper is presented in order to report an association of cat-scratch disease and an osteolytic lesion.

CASE REPORT

A 5-year-old white male was admitted to St. Christopher's Hospital for Children on Jan. 4, 1953, because of "a gland on his neck." Approximately four weeks before admission a swelling had developed in the right anterior cervical region at the angle of the mandible; this was treated symptomatically by local applications. Slight softening of the area was noted approximately two weeks later and the child was referred to another hospital for incision and drainage of the lesion. At the time of this procedure a biopsy was taken which was not diagnostic. Because of the subsequent development of pain in the region of the right hip, the child was referred here for further diagnosis and treatment.

When the child was admitted to this hospital, the parents stated that he had had slight fever at the onset of the present illness, but did not appear ill at any time. The lesion in the neck had gradually enlarged to the size of a "hen's egg" at the time that it was incised. About three weeks after the onset of cervical lymphadenopathy, the patient began to favor his right leg, and this latter complaint continued

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unabated until the present admission to the hospital. The parents had noted an indistinct swelling in the right groin of two or three weeks' duration, but reported no other swelling or complaint.

Past history was noncontributory except for the presence of frequent scratches from his cat without developing any definite local inflammatory lesions.

Physical examination revealed a well-developed and well-nourished child appearing neither acutely nor chronically ill. The rectal temperature was 100.6° F., pulse rate 92 per minute, respiratory rate 20 per minute, and the blood pressure was 124/80 mm. Hg. There was minimal injection of both tympanic membranes. A clear mucoid nasal discharge was present. There were dental caries. The pharynx was acutely injected. Immediately inferior to the angle of the

TABLE I

Hemoglobin	11.8 Gm. per cent
Red blood cells	4.12 million per cu.mm.
White blood cells	10,600 per cu.mm.
Segmented neutrophils	58 per cent
Nonsegmented neutrophils	4 per cent
Lymphocytes	27 per cent
Monocytes	6 per cent
Eosinophils	5 per cent
Platelets	516,000 per cu.mm.
Reticulocytes	0.6 per cent
Hematocrit	40
Erythrocyte sedimentation rate	18 mm./hour
Urinalysis	Normal
Heterophil antibody test	Negative
Tularemia agglutination	Negative
Typhoid O and H agglutination	Negative
Paratyphoid A and B agglutination	Negative
Brucella agglutination	Negative
Proteus OX_{19} agglutination	Negative
Complement fixation studies	
Syphilis	Negative
Lymphogranuloma	Negative
Blood chemical determinations	
Calcium	10.4 mg. per cent
Phosphorus	4.9 mg. per cent
Alkaline phosphatase	4.4 Bodansky Units
Total protein	6.2 Gm. per cent
A/G ratio	1.96
Cholesterol	344 mg. per cent
Skin tests	
PPD No. 1	Negative
PPD No. 2	Negative
Histoplasmin	Negative
Coccidioidin	Negative
Blastomycin	Negative
Haplosporangin	Negative
Frei test	Negative
Roentgenograms	
Chest and skeletal survey	Negative, except as noted in text

TABLE II. SKIN TESTS WITH CAT-SCRATCH ANTIGEN

Fessler	Induration 7 mm. with erythema 26 mm.
Campbell	Induration 1 cm. with surrounding erythema

colds, and the occurrence of a purulent right otitis media one month prior to the onset of the present illness. There had been no known ingestion of raw milk or contact with cattle, but there was a definite history of contact with cats, dogs, ducks, wild rabbits, and pheasants. He had received numerous

right mandible there was a healed incision with a dry crusted central sinus. The skin and subcutaneous tissue about the incision were firm and indurated but freely movable over the deeper structures; the area of induration measured approximately 2 × 4 cm. Several small discrete lymph nodes were present in the anterior cervical chains, bilaterally. A systolic murmur, interpreted as innocent, was heard along the left sternal border. There was definite tenderness in the right hip region when the right leg was hyperextended or flexed at the hip. There were soft nontender lymph nodes measuring 1 to 1.5 cm. in length in both inguinal regions. Small discrete, freely movable lymph nodes were present in both axillae and in the left epitrochlear area.

During the first nineteen days of hospitalization the patient's rectal temperature varied from 98° to 99.8° F. except for three occasions, at weekly intervals, when it rose abruptly to 101.2° F.

The laboratory tests performed during this hospitalization that proved to be negative or normal are summarized in Table I.

Roentgenographic studies of the right ilium revealed a radiolucent area 13.0 × 7.0 mm. along the margin of the right ilium. There was no evidence of fracture at the site of the lesion and no peripheral condensation of bone. The appearance suggested an eosinophilic granuloma of bone or a tuberculous or nonspecific osteomyelitis (Fig. 1).

Histologic examination of the material obtained at the time of the original biopsy* of the cervical lymph node revealed multiple granulomas with central necrosis and neutrophilic infiltrates surrounded by radially arranged large mononuclear cells; no foreign body nor Langhans' giant cells were identified, nor were fungi or bacteria identified in the hematoxylin and eosin preparations. The appearance of the node was interpreted as that of a granulomatous inflammation compatible with a variety of diseases, including cat-scratch fever (Fig. 2).

Surgical biopsy and culture of the lesion of the bone were performed; material taken from the area proved to be sterile on aerobic and anaerobic blood agar, Sabouraud's media, and Petragnani's media. Sections of the bone revealed subperiosteal formation of new bone. The marrow was replaced by loose vascular fibroblastic tissue infiltrated with small lymphocytes; occasional foci of neutrophils were present as well as areas of osseous destruction. Rare nodular aggregates of large mononuclear cells were present. The lesion was interpreted as a chronic osteomyelitis with a few questionable granulomatous foci (Fig. 3).

Fig. 1.—Right ilium. Note the osteolytic area below the anterior superior spine.

The intradermal injection in this patient of an antigen prepared from the lymph nodes of a child with cat-scratch disease (Campbell*) produced an area of induration 1 cm. in diameter with a surrounding erythema. This material produced no reaction when injected intradermally into two acutely ill children with lymphadenopathy, and into two convalescent children with lymphadenopathy. Intra-

*Sections of the original biopsy were supplied through the generosity of L. C. Pusch, M.D., York, Pennsylvania.

*Antigen supplied through the generosity of W. N. Campbell, M.D., Department of Pathology, Temple University School of Medicine.

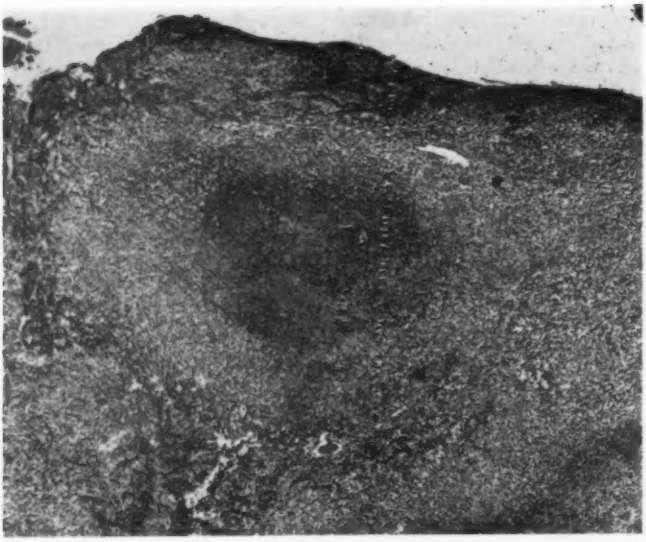

Fig. 2.—Cervical lymph node. Note the granuloma with central necrosis.

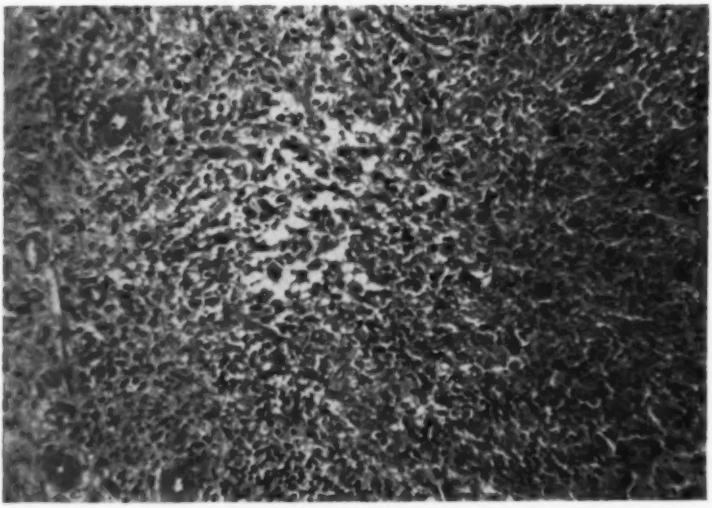

Fig. 3.—Ilium. Note the inflammatory infiltrate of lymphocytes and mononuclear cells, with some tendency of the latter to form a granulomatous lesion.

dermal injection of this antigen into the suspected cat produced no local response. Subsequently the Fessler* antigen was injected intradermally into the patient, and produced an area of induration 7 mm. in diameter with a surrounding erythema 26 mm. in diameter (Table II).

Fig. 4.—Note the complete healing of the previously described lesion.

During his period of hospitalization the patient steadily improved, and by the time of discharge on Feb. 2, 1953, he was asymptomatic. Roentgenograms of the lesion of the ilium demonstrated early healing. Physical examinations subsequently (Feb. 27, March 27, and May 22, 1953), have been normal except for persistent induration in the area of the cervical biopsy. Serial roentgenograms of the osseous lesion revealed complete healing by October 10, approximately nine months after the original x-ray (Fig. 4). All of the previously negative agglutination studies were again negative on Feb. 27, 1953.

DISCUSSION

Despite increased interest in this entity during the past few years the etiological agent has not been demonstrated. The diagnosis depends upon the clinical picture, a positive reaction to a skin test of undetermined specificity, plus microscopic demonstration of a granulomatous lesion. Intradermal tests in normal patients and in patients with cat-scratch disease, using antigens prepared from sterile, benign lymph nodes removed for a variety of reasons, might prove or disprove the specificity of the cat-scratch antigen used today. The development of a specific agglutination or complement fixation test must await the demonstration of the etiological agent.

The diagnosis of cat-scratch disease in this patient is based upon the clinical pattern, the histologic appearance of the cervical lymph node, and the presence of a positive skin test. Although the specificity of any of the observations may be questioned, such a combination of findings would appear to establish the diagnosis as conclusively as is possible at the present time. The relationship of the localized osteomyelitis in the ilium to the cat-scratch disease is certainly not established, and the processes may be independent ones. However, in view of the increasingly apparent protean nature of cat-scratch disease, the possibility that a causal relationship exists between this and the osteomyelitic lesion must be considered.

*Supplied through the generosity of W. B. Daniels, M.D., Washington, D. C.

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THE ACCURACY OF DIAGNOSIS OF BETA STREPTOCOCCAL INFECTIONS ON CLINICAL GROUNDS

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THE purpose of this study was to determine how accurate the physician (in private practice) can be in diagnosing beta hemolytic streptococcal throat infections in sick children on clinical grounds.

MATERIAL AND METHOD

Eleven hundred ninety-nine patients under 16 years of age whom we felt might have streptococcal infections were seen by either one of us during the period from Feb. 1 to Aug. 15, 1953. At the time the child was seen, data were obtained on age, sex, presence or absence of tonsils, time seen after onset, and symptoms and signs of disease. The symptoms listed were sore throat, whether it hurt to swallow, presence of headache, abdominal pain, vomiting, shaking chills, cough, hoarseness, and the degree of fever. The physical signs listed were the color of the throat, degree of exudate, presence of petechial hemorrhages in the throat, scarlatiniform rash, tender cervical glands, and infected ears. The white blood count when done was also noted. These data were checked on a McBee marginal punch card at the time the child was first seen. In one corner of the card we indicated at that time whether we thought that the infection was due to a streptococcal infection or not.

At the same visit a culture of the throat was obtained with a sterile cotton swab. In the office these swabs were streaked directly on sheep cell blood agar plates. In the home, the swabs were placed in sterile-stoppered test tubes containing sufficient infusion broth to keep them moist until they could be returned to the office and planted on blood agar plates. The plates were "read" from twelve to eighteen hours after planting. Suspicious colonies were isolated and stained by the Gram method. The result of the culture was then added to the card. Many of these isolated strains were subsequently sent to the Streptococcus Laboratory of the United States Public Health Service at Chamblee, Ga., for further study. With but few exceptions they were found to be Group A and of various types.

Children were considered to have a streptococcal infection only when beta hemolytic streptococcus was isolated from the throat culture. Since a positive culture was to be our only criterion of a streptococcal infection, it was necessary to determine how often a single culture would be negative when it should have been positive. An approximate estimate of this was obtained by taking duplicate cultures. In eighty-five cases in which one culture was positive, the second culture was also positive in 92 per cent of the cases. A second problem which is a corollary of the first, was to determine how often well

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children have streptococci in their throats. In order to answer this we took cultures of the throats of 150 normal, healthy children seen in the home or office during the period of the study. Only three (2 per cent) were found to be positive. From these studies we concluded that the use of a single culture as the criteria for streptococcal disease in the child seen in practice is valid, but has an error of approximately 10 per cent.

In making our clinical decision we used all information available to us at the time; symptoms, signs, knowledge of the current epidemiology, knowledge of previous infections in siblings when such had occurred, and leukocyte count when these had been done.

TABLE I

PREDICTION FROM CLINICAL FINDINGS	NO. OF CASES	CONFIRMATION BY CULTURE	PER CENT ACCURACY
Positive	495	372	75
Negative	704	540	77

TABLE II. THE FREQUENCY OF COMMON SYMPTOMS IN STREPTOCOCCAL AND NONSTREPTOCOCCAL INFECTIONS

SIGNS AND SYMPTOMS	STREP. % PRESENT	NONSTREP. % PRESENT	RATIO*
Sore throat	79	44	+2.0
Hurts to swallow	65	32	+2.0
Headache	51	34	+1.5
Abdominal pain	32	24	+1.3
Vomiting	33	18	+1.8
Chills	20	14	+1.4
Cough	15	19	-1.3
Hoarseness	4	6	-1.5

*Indicates the odds for (+) or against (-) a streptococcal infection.

TABLE III. APPEARANCE OF THROAT IN STREPTOCOCCAL AND NONSTREPTOCOCCAL INFECTIONS

APPEARANCE OF THROAT	STREP. % PRESENT	NONSTREP. % PRESENT	RATIO*
Normal	8	25	-3.1
Mildly inflamed	35	51	-1.5
Moderately inflamed	46	21	+2.8
Very inflamed	10	2	+5.0

*Indicates the odds for (+) or against (-) a streptococcal infection.

TABLE IV. DEGREE OF EXUDATE IN THROAT IN STREPTOCOCCAL AND NONSTREPTOCOCCAL INFECTIONS

AMOUNT EXUDATE	STREP. % PRESENT	NONSTREP. % PRESENT	RATIO*
None	46	73	- 1.6
Small	26	18	+ 1.4
Moderate	15	0.8	+18.8
Large	1	0.4	+ 2.5

*Indicates the odds for (+) or against (-) a streptococcal infection.

RESULTS

We found our percentage of accuracy in both positive and negative cultures to be approximately 75 per cent as shown in Table I.

There were certain symptoms that were more commonly seen in strepto-

coccal than in nonstreptococcal throat infections. Thus the complaints of "sore throat," "hurts to swallow," and vomiting (when present) occurred almost twice as often in streptococcal infections as in nonstreptococcal infections. The remainder of the symptoms and their frequency can be seen in Table II.

We had a total of 536 positive cultures; these were composed of positive cultures correctly predicted (372) and positive cultures incorrectly predicted (164). Thus expressed in another way, our accuracy in diagnosing streptococcal infections was 69.6 per cent.

The appearance of the throat as regards inflammation and exudate was helpful in arriving at the correct clinical diagnosis of streptococcal infections where these signs were moderate to severe in degree as can be seen from Tables III and IV.

Other signs were tabulated, namely, petechiae of throat, scarletlike rashes, clinically tender cervical glands, and inflamed ears, but they appeared too infrequently to be of much value diagnostically. Petechiae and scarlet rashes when present are very suggestive, but not diagnostic, of a streptococcal etiology.

In checking the age of the patient against our accuracy we found the older the child, the more often were we correct in our diagnosis (Table V). Incidentally, it is worth noting in this table the low incidence of Streptococci throat infections in children under 2 years of age.

We compared the diagnostic accuracy percentages of the streptococcal infection according to the time elapsed between the onset of the first symptom and the first examination; not surprisingly we found the highest percentage occurred when the children were examined twenty-four and seventy-two hours after the initial symptoms (Table VI).

TABLE V. FACTORS INFLUENCING ACCURACY OF DIAGNOSIS. AGE OF CASE VS. ACCURACY OF DIAGNOSIS

AGE IN YEARS	NUMBER POSITIVE	CORRECT PREDICTION (%)
0- 2	27	56
3- 5	165	73
6- 8	199	77
9-11	80	84
12 or more	24	88

TABLE VI. TIME SEEN AFTER ONSET VS. ACCURACY OF DIAGNOSIS

TIME SEEN	NUMBER POSITIVE	CORRECT PREDICTION (%)
12 hr.	122	66
24 hr.	157	82
36 hr.	68	87
48 hr.	42	83
3 da.	47	81
4 or more da.	36	58

White blood cell counts were obtained in eighty-four of streptococcal disease and 148 cases of nonstreptococcal infection. In general our conclusion was that white blood cell counts under 12,500 are uncommon in streptococcal infections (Table VII).

COMMENT

It is now generally conceded that both rheumatic fever and nephritis are initiated by beta streptococcal infections. Furthermore, Wannamaker and his associates[1] have given convincing evidence that rheumatic fever can be prevented by proper treatment of the initial streptococcal infection. In addition, unpublished data by Rammelkamp[2] show that acute glomerulonephritis may likewise be prevented.

If we are to prevent these complications, the proper diagnosis must be made with sufficient accuracy so that a minimum of cases go unrecognized. If the diagnosis of the primary streptococcal infection cannot be made on clinical grounds alone, it then is important that throat cultures be used diagnostically more generally in private practice and that facilities for evaluating them be made available to the practicing physician.

Our experience leads us to believe that throat cultures are necessary for accurate diagnosis. Many acute illnesses in youngsters are not seen more than once by the physician. It becomes increasingly easy to be correct in diagnosis with repeated visits, but such repeated visits are the exception rather than the rule. On a single visit we missed approximately 30 per cent of streptococcal infections. No single sign or symptom has high diagnostic value. Since we were particularly studying the problem we were on the alert for these infections and were constantly being checked by our culture results. Under ordinary conditions of practice we feel that our diagnostic ability might have been even less accurate. On numerous occasions a strongly positive throat culture was a surprise finding.

Without cultures one is forced into a much more widespread and frequently unnecessary use of penicillin; even then many cases will be missed and not be treated properly. Although not covered in this study, the response to penicillin of streptococcal infections is dramatic and is of high diagnostic value. Our experience has led us to believe that unless marked improvement and near normal temperature readings are not attained within twenty-four hours after penicillin, the disease in question is not streptococcal in origin.

Therefore the diagnosis of the infection is most important, and we feel that throat cultures are necessary for accuracy.

TABLE VII. THE WHITE BLOOD COUNT IN STREPTOCOCCAL AND NONSTREPTOCOCCAL INFECTIONS

WBC IN THOUSANDS	STREP.		NONSTREP.	
	NUMBER	% TOTAL	NUMBER	% TOTAL
Under 10	5	5.9	36	24.3
10-12.4	3	3.6	21	14.2
12.5-14.9	11	13.1	16	10.8
15.0-17.4	19	22.6	17	11.5
17.5-19.9	10	11.9	22	14.8
20.0-22.4	14	16.7	14	9.5
22.5-24.9	11	13.1	6	4.1
25 or above	11	13.1	16	10.8
Total	84	100.0	148	100.0

SUMMARY

In a group of 1,199 cases of illness in children suspected of having streptococcal infection a diagnosis on clinical grounds was correct in approximately 75 per cent of patients. Certain symptoms and signs were suggestive of streptococcal infection but none were diagnostic. Throat cultures materially increase the accuracy of diagnosis.

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OBSERVATIONS ON THE PHYSIQUE AND GROWTH OF CHILDREN WITH CONGENITAL HEART DISEASE

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THE physique and growth of children with congenital heart disease have not been the subject of frequent investigation. Most articles on the subject of congenital heart disease have dealt with the problem only in general terms, and for the most part the comments have not been the result of careful analysis of height and weight data. Three previous communications from our laboratory have dealt with the problem in patients with patent ductus arteriosus,[1] tetralogy of Fallot,[2] and pulmonary stenosis without cyanosis.[3] The purpose of this report is to summarize our previous work and to add to it observations on patients with interatrial septal defect, interventricular septal defect, and coarctation of the aorta. As the result of these observations, certain conclusions seem justified.

CLINICAL MATERIAL AND METHODS

All of the 229 children reported in this study were seen at the University of Minnesota Hospitals or at the U.C.L.A. Medical Center. In most, the clinical diagnosis was confirmed at surgery, and in those not operated upon it was based upon the clinical findings and data obtained during right heart catheterization.

The Wetzel "Grid for Evaluating Fitness"[4] was used for interpreting the height, weight, and age data because of the comprehensive picture one obtains by its use. The criteria for satisfactory growth, recommended by Wetzel, were used by us in interpreting the data. The tolerance limits were ½ channel deviation per ten levels of progress, and two to three levels from the patient's own auxodrome in any one year. These later criteria were used only in evaluating the patients with patent ductus arteriosus and tetralogy of Fallot since we did not have enough observations in the other conditions to make the conclusions valid on this point.

PATENT DUCTUS ARTERIOSUS

The growth data on sixty-one children over the age of 5 years with a patent ductus arteriosus were analyzed. In thirty-one of these sixty-one patients, at least two sets of height and weight data, taken a minimum of six months apart, were available preoperatively. The lowest auxodrome and the lowest channel reached on the Wetzel Grid preoperatively by each of these thirty-one patients are tabulated in Table I. It can be seen that for the most part both the physique and rate of growth are normal. There are a few more than would normally be expected

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with thin physiques and slow rates of growth. Using Wetzel's criteria for satisfactory growth as seen in Table II, one patient had a gain in physique and one a gain in rate of growth preoperatively. Six patients, however, experienced a loss in physique, and two a loss in rate of growth.

Height and weight data were available on only fifty-three of the sixty-one patients postoperatively; twenty-three of these patients were ones in whom preoperative data were also available. In all instances one set of these data was obtained at least one year after surgery. As shown in Table III, nineteen of the fifty-three exhibited, postoperatively, a gain in both physique and rate of growth, but eight patients had a loss in physique, and six had a loss in rate of growth.

TABLE I. PATENT DUCTUS ARTERIOSUS
PREOPERATIVE PHYSIQUE AND RATE OF GROWTH

Lowest channel	A_4	A_3A_2	A_1MB_1	B_2	B_3	B_4	Total No. of Patients
No. of patients	0	5	15	6	1	4	31
Lowest auxodrome	0-2%	2-15%	15-67%	67-82%	82-98%	98-100%	
No. of patients	1	2	10	8	4	6	31

TABLE II. PATENT DUCTUS ARTERIOSUS
EVALUATION OF PREOPERATIVE PHYSIQUE AND RATE OF GROWTH*

	GAIN	SATISFACTORY	LOSS	TOTAL NO. OF PATIENTS
Physique	1	24	6	31
Rate of growth	1	28	2	31

*Tolerance limits used were ½ channel deviation per ten levels of progress and two to three levels from the patient's own auxodrome in any one year.

TABLE III. PATENT DUCTUS ARTERIOSUS
EVALUATION OF POSTOPERATIVE PHYSIQUE AND RATE OF GROWTH

	GAIN	SATISFACTORY	LOSS	TOTAL NO. OF PATIENTS
Physique	19	26	8	53
Rate of growth	19	28	6	53

TETRALOGY OF FALLOT

The growth data on sixty-six children with tetralogy of Fallot were analyzed. The same criteria were applied to these patients as were applied to those with patent ductus arteriosus. As seen in Table IV, the physique channel and the rate of growth preoperatively were not too unlike those found in patients with patent ductus arteriosus, i.e., a majority had normal physiques and rates of growth.

Sufficient data were available on forty-seven of the sixty-six patients operated upon to evaluate the effect of surgery. As seen in Table V, twelve patients showed a gain in physique, and fourten showed a gain in rate of growth. On the other hand, twenty patients had a loss in physique, and twenty-one had a loss in rate of growth. Among those patients operated upon there appeared to be no correlation between the success of the operation (as measured by increase in exercise tolerance) and the changes in growth.

INTERATRIAL SEPTAL DEFECT

Twenty-two infants and children with an interatrial septal defect were available for study. Sixteen of these

were old enough that their heights and weights could be plotted on the older grid of Wetzel's; the other six had to be plotted on the Infant Grid. As seen in Table VI, the older child with an interatrial septal defect had either an average physique or slightly thinner than average physique; none were obese or stocky. The rate of growth of these same children was either average or slower than average; none were accelerated in their growth.

The data on the infants were too small to put into table form. In brief, it might be stated that the physiques of the infants were fairly normal but in every instance the rate of growth was slow. This could be the result of selection since probably only those infants with interatrial septal defects that were large enough to produce symptoms would be studied at such an early age. Accordingly these same infants would be the ones with moderate to extreme pulmonary congestion which makes the patient more prone to develop respiratory disease and secondary growth retardation.

TABLE IV. TETRALOGY OF FALLOT
PREOPERATIVE PHYSIQUE AND RATE OF GROWTH

Lowest channel	A_1	A_2A_2	A_1MB_1	B_2	B_3	B_4	Total No. of Patients
No. of patients	4	4	26	19	9	4	66
Lowest auxodrome	0-2%	2-15%	15-67%	67-82%	82-98%	98-100%	
No. of patients	1	3	12	25	13	12	66

TABLE V. TETRALOGY OF FALLOT
EVALUATION OF POSTOPERATIVE PHYSIQUE AND RATE OF GROWTH

	GAIN	SATISFACTORY	LOSS	TOTAL NO. OF PATIENTS
Physique	12	15	20	47
Rate of growth	14	12	21	47

TABLE VI. INTERATRIAL SEPTAL DEFECT
PHYSIQUE AND RATE OF GROWTH

Lowest channel	A_1	A_2A_2	A_1MB_1	B_2	B_3	B_4	Total No. of Patients
No. of patients	0	1	7	5	3	0	16
Lowest auxodrome	0-2%	2-15%	15-67%	67-82%	82-98%	98-100%	
No. of patients	0	0	8	3	3	2	16

TABLE VII. INTERVENTRICULAR SEPTAL DEFECT
PHYSIQUE AND RATE OF GROWTH

Lowest channel	A_1	A_2A_2	A_1MB_1	B_2	B_3	B_4	Total No. of Patients
No. of patients	0	0	12	5	2	3	22
Lowest auxodrome	0-2%	2-15%	15-67%	67-82%	82-98%	98-100%	
No. of patients	0	1	9	7	3	2	22

INTERVENTRICULAR SEPTAL DEFECT

Thirty-five infants and children with an interventricular septal defect were available for study. Twenty-two of these were old enough that their heights and weights could be plotted on the older Wetzel Grid; the other thirteen had to be plotted on the Infant Grid. Table VII summarizes the findings of the twenty-two children on the older Wetzel Grid. The data ob-

tained here were similar to those obtained from the patients with a patent ductus arteriosus, and interatrial septal defect. For the most part the physiques were average or slightly thinner than average, and the rates of growth were average or slightly slower than average. Many, however, had entirely normal growth.

As in the case of infants with an interatrial septal defect, the majority of the thirteen infants with an interventricular septal defect had fairly normal physiques. With the exception of two of the patients, the infants had a slow rate of growth; however, the cause for this slow rate of growth may have been the same as in the infants with interatrial septal defect; i.e., selected cases with large shunts sufficient to make the patient more prone to respiratory disease and secondary growth failure. The following brief case history illustrates this last point.

P. S., a female infant, was known to have congenital heart disease at birth. For the first twenty-one months of her life she was admitted numerous times to the Minneapolis General Hospital for recurrent respiratory disease, including pneumonia. At least one-half of the twenty-one months was spent in the hospital. At 14 months of age she weighed 17 pounds and was 30 inches tall; at 21 months she weighed 20 pounds and was 32 inches tall; both points showed poor growth. At 21 months of age she was started on prophylactic sulfadiazine each day, and concomitant with this the frequency and severity of respiratory infections decreased. By 45 months of age she weighed 32½ pounds and was 39½ inches tall (a normal physique and rate of growth for a child her age). The interventricular septal defect in this case was conclusively demonstrated when the child was 32 months of age; moderate pulmonary hypertension was also present.

COARCTATION OF THE AORTA

Twenty-seven infants and children with a coarctation of the aorta were available for study. Twenty-one of these were old enough that their heights and weights could be plotted on the older Wetzel Grid, the other six had to be plotted on the Infant Grid. As seen in Table VIII, the older child with a coarctation of the aorta in general had preoperatively a normal physique and a normal rate of growth.

TABLE VIII. COARCTATION OF THE AORTA
PHYSIQUE AND RATE OF GROWTH

Lowest channel	A_4	A_3A_2	A_1MB_1	B_2	B_3	B_4	Total No. of Patients
No. of patients	1	3	12	3	0	2	21
Lowest auxodrome	0-2%	2-15%	15-67%	67-82%	82-98%	98-100%	
No. of patients	0	5	9	4	2	1	21

The data on the six infants were too small to put into table form. However, it might be stated that in those patients who had episodes of heart failure, growth retardation was present. However, those infants without heart failure had a normal rate of growth.

Twelve of the twenty-seven infants and children with coarctation of the aorta had a surgical resection of the coarcted segment. The effect of the surgery on the growth of these patients was negligible in terms of producing change in either the physique or the rate of growth.

PULMONARY STENOSIS WITHOUT CYANOSIS

Data were available on eighteen patients (who were 5 years of age or older) with pulmonary stenosis without cyanosis. As seen in Table IX, the points of the majority of these patients fell into the A_1, M, and B_1 channels and the speed of growth appeared to be close to normal. None of these patients had arterial desaturation. All of the patients were operated upon, at which time a pulmonary valvulotomy was done. The follow-up period on these patients, however, has been too short for conclusions to be drawn as to the effect of such surgery on the growth.

Table IX. Pulmonary Stenosis Without Cyanosis
Physique and Rate of Growth

Lowest channel	A_1	A_1A_2	A_1MB_1	B_2	B_3	B_4	Total No. of Patients
No. of patients	0	1	15	1	1	0	18
Lowest auxodrome	0-2%	2-15%	15-67%	67-82%	82-98%	98-100%	
No. of patients	0	1	4	9	2	2	18

DISCUSSION

From these observations it might be concluded justifiably that patients with congenital heart disease do not *necessarily* have abnormal growth either in terms of physique or rate of growth. That children with large left-to-right shunts can have impaired growth is acknowledged. It is our opinion, however, that such growth retardation may be the result of the increased incidence of respiratory disease which is promoted by the pulmonary congestion secondary to the left-to-right shunt. No conclusive data on this point are available at the present time, however. When such infection is minimized, maximum growth for the individual patient may occur in spite of the large left-to-right shunt. This point is illustrated by the case of C. W., a 9-year-old female, with a *patent ductus arteriosus*, demonstrated both by right heart catheterization studies and surgical exploration, and who had minimal respiratory disease. The left-to-right shunt through the patent ductus arteriosus in her case was of the magnitude of 66 per cent of the left ventricular output when calculated according to the Fick principle, the estimated pulmonary blood flow being approximately 16 liters per minute at rest. In spite of this large shunt, at 8 years 10 months of age she weighed 60½ pounds and was 51 inches tall. This placed her in the M channel and between the 15 and 67 per cent auxodromes on the Wetzel Grid. Following surgery her growth remained unchanged except for the increased rate of growth associated with adolescence.

It is also our opinion that the *anoxemia* present in children with cyanotic heart disease does not produce growth retardation per se. If such were the case one might expect:

1. The greatest growth retardation in patients with the severest anoxemia.
2. Regular improvement in growth following successful surgery for cyanotic heart disease.

The observations just reported have shown that such is not the case. In fact some of the best surgical results were accompanied by the poorest growth, and likewise some of the children with the severest anoxemia had

normal growth. This last point is illustrated by infant K. T., who had an arterial oxygen saturation of less than 20 per cent and was having nine to twelve fainting episodes a day. In spite of this he weighed 22 pounds and was 30 inches long at 10 months of age. This patient had a tetralogy of Fallot of the pseudotruncus type.

Comment should also be made regarding the growth of children with coaretation of the aorta. Most textbooks refer to the classic appearance of the patient, particularly boys. One author states[5] that "Boys and men with this malformation are usually large, s t r a p p i n g, well-developed, sturdy individuals. Indeed, the full physical development and the early onset of puberty are quite as distinctive features of this malformation as are the poor physical development and delayed onset of puberty in patients with an auricular septal defect." From our data one must conclude that the over-all physique and the rate of growth of children with coaretation of the aorta is not unusual and in fact is not too dissimilar from those patients with interatrial septal defects. On the other hand, it is entirely possible that in patients with coaretation of the aorta the physical changes in the upper trunk and extremities are so offset by changes in the lower that they counterbalance each other. Thus when the total physique is considered it falls within the normal range.

Some patients with obvious congenital heart disease have been observed by us to have marked growth retardation. In some instances such patients have had a serious cardiac lesion placing a great strain on the heart. In other instances, however, clinical and physiological studies have demonstrated negligible deviation from the normal state. It was thought that in the latter instance, the growth retardation was *primary* (due to hereditary or in utero factors), or, if secondary, secondary to some *other* cause but not due to the heart lesion per se. In such instances the left-to-right shunt was too small to produce symptoms or growth retardation.

Children with congenital heart disease are also subject to the other causes of growth failure or acceleration as are the rest of the population. These, therefore, must be considered in each case. Infectious processes, metabolic derangement, nutritional inadequacy, and emotional disturbances must all be considered. In congenital heart disease particularly it is not uncommon to see emotional problems arise in children as a result of the overprotection and anxiety of the parents.

SUMMARY

1. Data regarding the growth of 229 children with certain congenital heart lesions have been reviewed, using the Wetzel Grid technique. The lesions studied were patent ductus arteriosus, tetralogy of Fallot, interatrial septal defect, interventricular septal defect, coaretation of the aorta, and pulmonary stenosis without cyanosis.

2. In general it can be concluded that patients with congenital heart disease do not *necessarily* have abnormal growth either in terms of physique or rate of growth.

3. If a large left-to-right shunt is present the resultant pulmonary congestion may predispose the patient to respiratory disease which in turn can produce growth retardation. If such

infection is minimized, maximum growth may occur in spite of the large left-to-right shunt.

4. Patients possessing congenital heart lesions with anoxemia such as tetralogy of Fallot do not necessarily have growth retardation, and surgery performed to decrease the anoxemia is not necessarily followed by improved growth.

5. In some patients with congenital heart disease, growth retardation is marked, but clinical and physiological studies show little deviation from the normal state. In such instances, the growth retardation is thought to be primarily due to genetic or in utero factors, or, if secondary, secondary to something other than the heart lesion itself. Emotional disturbances can be an important secondary cause of poor growth in this regard.

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SOME CLINICAL ASPECTS OF TYPHOID FEVER IN INFANCY

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PAGO PAGO, AMERICAN SAMOA

TYPHOID fever is a disease that is usually considered rare in infancy and is practically nonexistent in any age group where rigid sanitary rules are enforced.[1] In American Samoa, where sanitation is practically nil, twenty-seven out of thirty village water supplies were found to be positive for typhoid bacillus. The entire main island is mountainous with only a small amount of land that can be used for cultivation. As a rule, this land is located on the slopes of the mountain above the water supply.

Thirty infants between the ages of 5 weeks and 11 months have been admitted to the hospital, twenty-one males and nine females. In two of the thirty cases, the onset of the illness was two weeks prior to hospital admission. One of these infants was admitted because of weight loss and the other because of irregular bouts of fever. In the remaining cases, the onset of the illness was one to seven days prior to admission. Usually the more explosive the onset, the shorter the period prior to hospitalization. The chief complaints as stated by the mothers are shown in Table I.

TABLE I. CHIEF COMPLAINTS

	NO. OF CASES	PER CENT
Vomiting and diarrhea	9	30
Diarrhea only	6	20
Vomiting only	4	13
Cough	4	13
Fever	3	10
Convulsions	2	7
Anorexia, weight loss	2	7

TABLE II. RESPIRATORY MANIFESTATIONS

	NO. OF CASES	PER CENT
Râles	12	40
Cough	10	33
Cyanosis	4	13
Coryza	2	7
Otitis media	1	3
Epistaxis	0	0

The diagnosis of typhoid was made on routine stool cultures on admission. The respiratory manifestations are shown in Table II, the gastrointestinal manifestations in Table III, and the complications in Table IV.

TABLE III. GASTROINTESTINAL MANIFESTATIONS

	NO. OF CASES	PER CENT
Dehydration	10	33
Vomiting and diarrhea	9	30
Tympanitis	9	30
Diarrhea only	6	20
Vomiting only	4	13
Palpable spleen	4	13
Palpable liver	3	10
Anorexia, weight loss	2	7
Rose spots	0	0

TABLE IV. COMPLICATIONS

	NO. OF CASES	PER CENT
Bronchial pneumonia	10	33
Chronic malnutrition	7	23
Typhoid meningitis	1	3
Typhoid carrier	1	3

DISCUSSION

The stool cultures were positive in all cases on admission and in one case on discharge. Twenty-four patients had a leukocytosis of over 10,000 and one a leukopenia (this patient later expired). The stools became negative in from four to twenty-four days, aver-

From Department of Pediatrics, Public Health Service.

aging 11.7 days, except in the case of the carrier. In this case, the stools were positive six months after discharge. The temperature returned to normal in from two to seven days, averaging four days, except in the one patient who later relapsed and died. In this case, the temperature returned to normal after ten days of the first admission. Of the thirty infants, two relapsed, one after forty-eight days and the other after fifty-seven days. In the case of the infant who relapsed after fifty-seven days and showed the leukopenia on admission, this patient was readmitted to the hospital and died on the fourth hospital day from severe malnutrition and typhoid fever (the stools having again become positive). The other patient who relapsed remained in the hospital for sixteen days on the second admission. The second infant who expired died in the emergency room before he could be admitted to the hospital.

The treatment consisted of subcutaneous fluids to correct dehydration, using 2½ per cent glucose in saline and ⅙ molar sodium lactate. Whole milk and a general infant diet for age was given to all patients as soon as the vomiting was controlled. Aureomycin alone was used in seven infants; however, the dosage recommended by the manufacturer was found to be too small. It was also used in three other cases prior to the use of chloramphenicol. The dosage was 40 to 90 mg. per pound for the total twenty-four-hour dose and was given in four to six doses for a period of five to eight days. It was stopped in one case because of vomiting. Chloramphenicol was given to the remaining twenty-three infants in doses of 60 to 120 mg. per pound for the total twenty-four-hour dose, the dosage being given every four hours for seven to eleven days. The infant who was discharged as a carrier received 120 mg. per pound for the total twenty-four-hour dose for a period of ten days. Aureomycin and chloramphenicol were both used on the two infants who relapsed and on the infant discharged as a carrier. None of the patients receiving either Aureomycin or chloramphenicol developed a granulocytopenia. Seven infants were readmitted because of malnutrition. The stools of all have remained negative except for the two infants who relapsed and the one who is a carrier.

The only method to date which has shown any indications of controlling typhoid has been mass immunizations, as the mothers have shown a complete indifference to boiling water. Newborn babies are given ⅛ c.c. of triple typhoid vaccine twenty-four hours prior to discharge from the hospital and three subsequent injections of ¼ c.c. at weekly intervals. To date no untoward effects have been noted other than a slight restlessness the night following the second or third injection. Temperatures have been checked at four-hour intervals following the first and second injections and there have been no reported elevations.

SUMMARY

1. Thirty cases of typhoid in infants are reported.

2. There were two relapses, two deaths and one patient was discharged as a carrier (the stools still being positive after six months).

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CARDIAC INVOLVEMENT IN PROGRESSIVE MUSCULAR DYSTROPHY

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THIRTY years after the inital clinical description of progressive muscular dystrophy[1] appeared the first mention[2] of cardiac involvement. Thereafter, reports[3-17] of myocardial changes followed with increasing frequency. The incidence of this complication is still unknown although a figure of 50 per cent in a series of thirty-three patients has been noted.[16] Because myocardial degeneration in progressive muscular dystrophy may be unfamiliar to many pediatricians, an example of this association is reported here.

CASE REPORT

F. H., a 13-year-old white male, was admitted through the courtesy of Dr. Ira A. Manville to Doernbecher Memorial Hospital for Children on July 16, 1952. (A maternal uncle and two younger brothers have progressive muscular dystrophy.)

At the age of 7 years he began to climb stairs with difficulty and to stumble occasionally when walking. Shortly thereafter he could arise from a recumbent position only by "climbing up his legs" and his calf muscles became enlarged. Later his arms showed progressive involvement. For two years prior to admission he was unable to walk. A year before admission he became severely dyspneic and thereafter digitalization was maintained sporadically. Two weeks before hospitalization there appeared ankle and pretibial edema, hepatic enlargement, and pulmonary congestion. Treatment with digitalis and diuretics induced loss of edema and relief of dyspnea.

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On physical examination he was an obese male with pronounced tongue enlargement, extreme atrophy of both legs, moderate atrophy of both arms, and multiple flexion contractures. Blood pressure was 110/70. Cardiac rhythm and rate were regular although he had a labile sinus tachycardia (84-126 beats per minute). No murmurs could be heard and both lung fields were clear. No gross hepatosplenomegaly was found.

Laboratory determinations included a hemoglobin of 15.6 Gm. per cent, 5.2 million red blood cells, 4,800 white blood cells (normal differential), and normal urinalysis. X-ray examination (Fig. 1) demonstrated moderate left auricular enlargement with diffuse enlargement of the entire heart. Lung fields were considered normal. An electrocardiogram (Fig. 2) was abnormal with minimal splintering of the QRS complex and left axis deviation. A P-R interval of 0.16 second and a QRS duration of 0.08 were noted. (Digitalization had been maintained for two weeks prior to electrocardiogram recording.)

During two weeks of hospitalization no medications were given and no evidence of cardiac decompensation developed. One week after discharge pitting edema of both lower legs was found although the lungs were clear and the liver impalpable. An intramuscular injection of mercurial diuretic was given and a daily regimen of oral diuretics instituted. Four days later he developed pulmonary edema and expired.

Post-mortem Examination.—There was pronounced general replacement of body musculature with fat; a section of the left biceps femoris contained little recognizable muscle tissue. The liver extended 4 cm. below the right costal margin and its cut surface

Fig. 1.—X-ray of the chest, showing diffuse cardiac enlargement as well as the double density of right auricular enlargement.

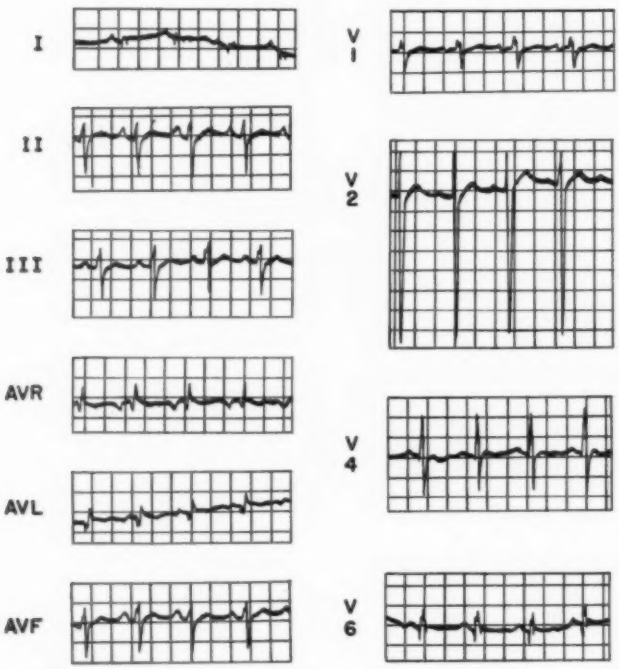
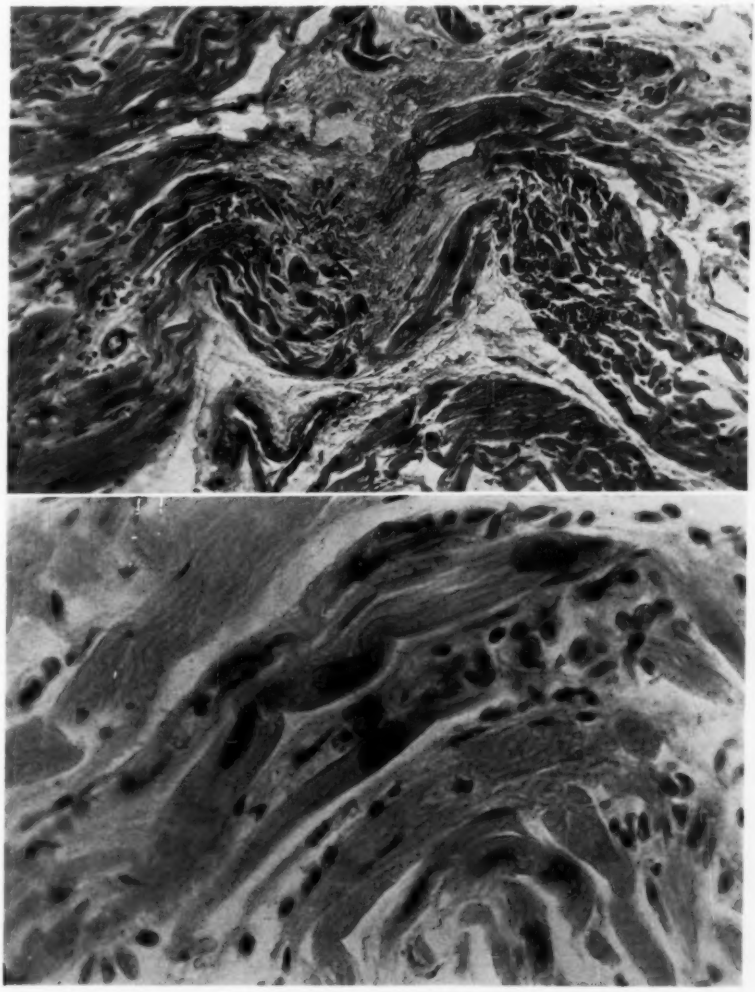


Fig. 2.—Electrocardiogram tracing demonstrating left axis deviation and notching of QRS complex.

presented the appearance of chronic passive congestion. Both lungs contained abundant frothy brownish fluid; a hemorrhagic infarct was found in the right lower lobe.

The heart was globular, measured 13 cm. in transverse diameter, and weighed 460 grams. Both ventricles were greatly dilated; the right ventricular wall measured 5 mm. in thickness, the left 1.5 cm. On microscopic study (Fig. 3, *A* and *B*) there were numerous areas in the myocardium where fibrous tissue totally replaced muscle. In others there was degeneration of muscle with some loss of striation, diminution, and distortion of fibers and proliferation of sarcolemmal and inflammatory nuclei. Sections of skeletal muscle (Fig. 4, *A* and *B*) contained essentially the same changes seen in the cardiac fibers.



A.

B.

Fig. 3.—Degeneration of muscle and replacement by fibrous tissue. Proliferation of sarcolemmal and inflammatory nuclei. *A*, Low power; *B*, high power. (×660; reduced ⅓.)

DISCUSSION

Our patient demonstrated many of the clinical findings previously recorded among thirty-two children with sleeping pulses of 89 and 77, respectively, in seven patients with muscular dystrophy; it also disclosed an excessive lability of pulse. Arrhythmias

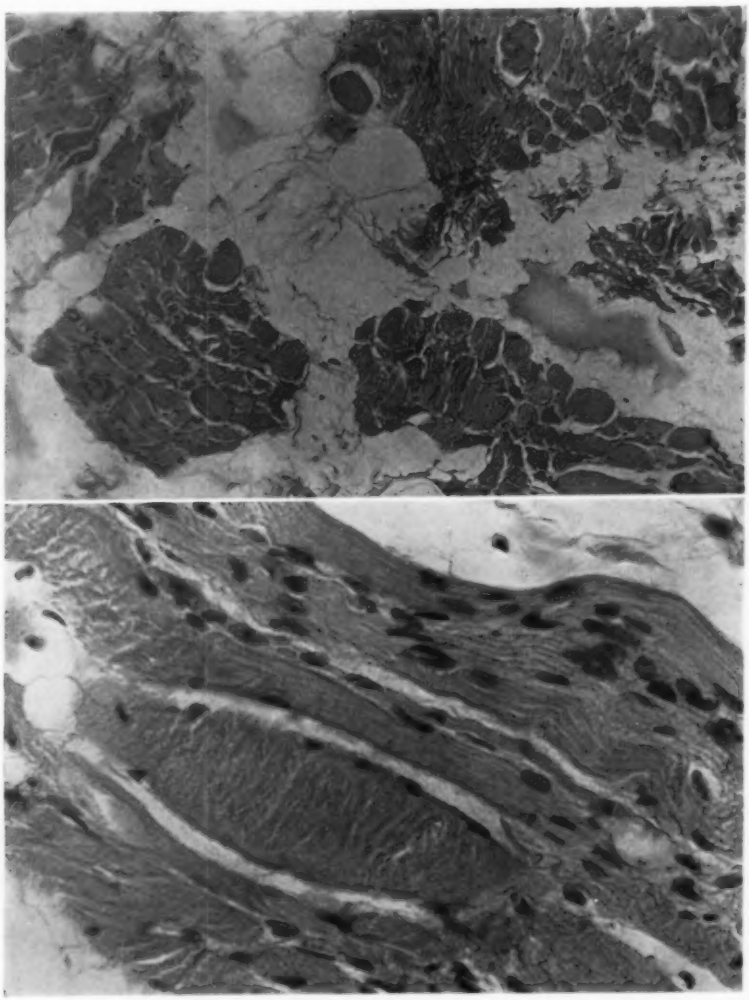


Fig. 4.—Skeletal muscle. Borderline hypertrophy and advanced atrophy of fibers in same field. Some proliferation of sarcolemmal nuclei. *A*, Low power; *B*, high power. (×660; reduced ⅕.)

dystrophic myocardial involvement. Tachycardia has been the earliest and most consistent sign. A previous study[7] found average waking and with palpitations and periods of weakness may be associated with the tachycardia. In only three children[9, 14] have blood pressure values been re-

ported and all were normal. Normal heart sounds have been usual although occasionally the first sound was slurred[10] or feeble.[7] Such murmurs as have been reported[10, 11, 13, 16, 17] were usually systolic, soft, and apical.

In most instances, x-rays of the heart have been normal. However, two patients in congestive failure[12] had pronounced dilatation and another[11] in apparent compensation had diffuse cardiac enlargement. A multitude of electrocardiographic changes have been noted, all indicative of diffuse myocardial damage. The P-R interval has been normal; the axis deviation may be either right or left. In one instance bundle branch block was discovered.[13]

As in this patient, classic signs of cardiac decompensation have been encountered,[10-13, 15] and in two instances[10] acute failure resulted in death. The remainder responded to digitalis and diuretic therapy. Cessation of treatment in two patients,[12] as in ours, resulted in return of decompensation; restitution was followed by improvement.

Records of post-mortem heart muscle studies have all shown pale, flabby, friable muscle, atrophy and hypertrophy of muscle fibers, and increased interstitial fibrous tissue.[2-6, 8, 11, 14, 17]

SUMMARY

1. A child with progressive muscular dystrophy and cardiac muscle involvement has been presented.

2. Clinical manifestations in children as reported in the literature have been summarized.

I am indebted to Dr. Nelson R. Niles, Department of Pathology, for his selection of pathological illustrations, and to Dr. Allan J. Hill, Professor of Pediatrics, for his guidance.

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VARIATIONS IN CLINICAL MANIFESTATIONS OF OSTEOPETROSIS

Report of Two Cases

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OSTEOPETROSIS is a rare developmental disease of bone that varies widely in clinical manifestations, involving other organ systems. The diagnosis is made roentgenologically because of typical bone changes.

Former reports have divided the disease into benign and malignant forms.[1, 2] The two cases we are reporting represent these types: one simulating acute leukemia, the other, malnutrition and retardation of growth and development.

CASE REPORTS

Case 1.—J. T., a white female, aged 4 months, was transferred from another hospital to the pediatric wards of University Hospital on April 19, 1949, with a tentative diagnosis of acute leukemia with extensive leukemic infiltration of bone.

Past history revealed a full-term spontaneous delivery with birth weight of 2,950 grams. There was moderate toxemia of pregnancy in the eighth month. The infant was formula-fed and received a multiple-vitamin preparation.

Family history revealed no evidence of consanguinity nor symptomatology compatible with osteopetrosis in the last three generations. The father and mother are of Italian descent and in good health. One previous pregnancy terminated in a miscarriage. The maternal grandmother's first two pregnancies also terminated in miscarriages, causes unknown.

From the pediatric services of University and Gouverneur Hospitals.

The child was well until 2½ months of age, when, concomitant to an infection of the upper respiratory tract with diarrhea, the parents noted "searching" movements of the eyes. She was admitted to a local hospital where physical findings of significance were: aimless roving of the eyes, fixed pupils, nystagmus, optic atrophy, and hepato-splenomegaly. Laboratory data (furnished by transcript): hemoglobin 8.0 Gm. per 100 c.c., red blood count 2.4 million per cubic millimeter, white blood count 18,800 per cubic millimeter with 35 per cent neutrophils, 52 per cent lymphocytes, 4 per cent monocytes, 3 per cent eosinophils, 6 per cent basophils, and 19 nucleated red cells per 100 white cells. Reticulocyte count was 11.6 per cent; platelets, 37,000 per cubic millimeter. Attempts at bone marrow aspiration were unsuccessful. Roentgenographic examination of the entire skeleton revealed increased density of the bones of the base of the skull and axial skeleton. There were transverse zones of mottled rarefaction at the ends of the long bones, especially the distal radius, ulna and femur, and proximal tibia. Epiphyseal centers were compatible with age but appeared mottled. Generalized periosteal new bone was seen along the diaphysis of the long bones. Diagnosis was acute leukemia with leukemic infiltration of bone. The child received a blood transfusion and was transferred to University Hospital for further study.

On admission to our service at 4 months of age the child weighed 4,200 grams. Physical findings were unchanged. Length 58.5 cm., head circumference 41.3 cm., chest circum-

Fig. 1.—Case 1. Patient at 22 months of age. Note the heavy ring of bone around the orbits.

ference 35.6 cm. Initial laboratory data: sedimentation rate (Westergren) 13 mm. per hour, hemoglobin 10.2 Gm. per 100 c.c., red blood count 3.5 million per cubic millimeter, white blood count 19,550 per cubic millimeter with 20 per cent neutrophils, 59 per cent lymphocytes, 15 per cent eosinophils, 1 per cent basophils, 15 per cent monocytes, 2 per cent myelocytes, 5 normoblasts per 100 white cells. The peripheral smear revealed moderate anisocytosis and poikilocytosis but no sickle or target cells. Reticulocyte count was 1.8 per cent; platelets, 100,000 per cubic millimeter. Bleeding time (Duke) was 15 minutes. Capillary clotting time was 4 minutes. Nonprotein nitrogen was 39.5 mg.; total protein, 5.5 Gm.; true blood glucose, 40 mg. Prothrombin time was 15.5 sec. (normal 14 to 16 sec.). Complement fixation test for syphilis was negative. Urinalysis and spinal fluid examinations were within normal limits. Bone marrow aspirations revealed myelocytic hyperplasia of marrow. Repeated roentgenograms (Figs. 1, 2, 3) were reported as showing increased structural density of all bones, especially of the base of the skull and the orbits. There were transverse linear defects at the metaphyseal ends of the long bones. The diagnosis of osteopetrosis was then definitely established.

The child was discharged but returned for blood transfusions every six to eight weeks. She gained little weight and appeared unusually susceptible to upper respiratory infections. All four incisors erupted at 10 months of age, but the enamel was mottled and discolored. She walked with support at 15 months. Subsequent blood counts showed a persistence of and increasing anemia with moderate leukocytosis. Up to 120 normoblasts, 3 to 7 myelocytes, and 1 to 3 myeloblasts per 100 white cells were seen on peripheral smear. Plate-

Fig. 2.—Case 1. Patient at one year of age. Lower extremities, showing characteristic clubbing and diffuse density of long bones with loss of normal architecture.

lets ranged from 50,000 to 100,000 per cubic millimeter, and bleeding time was always prolonged.

At 22 months of age the child was admitted for a trial of ACTH therapy. Weight was 10 kilograms, length 77.5 cm., head circumference 49.5 cm., chest 47 cm. Spleen and liver were larger, and, for the first time, generalized discrete lymphadenopathy was noted. A soft apical systolic murmur was heard. Thorn test, with 15 mg. of ACTH, dropped the eosinophils from 340 to 77 per cubic millimeter in four hours.

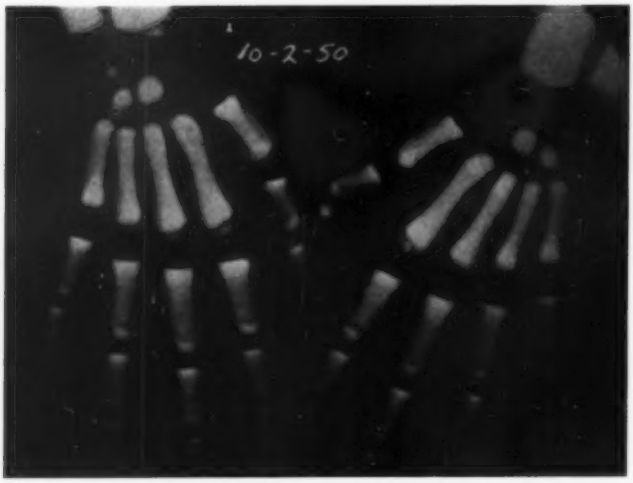

Fig. 3.—Case 1. Patient at 22 months of age. Excessive density and fanlike appearance of the growing ends of the metacarpals and phalanges. Three carpal centers are present.

Total serum calcium was 11.6 mg.; serum inorganic phosphorus, 4.3 mg.; alkaline phosphatase, 2.6 and 4.2 Bodanski units; acid phosphatase, 19.2 and 14.3 Gutman units (normal 0.5 to 2.5)[3]; thymol turbidity, 2 units; total serum cholesterol, 155 mg. Serum sodium and potassium were within normal limits. Urine Sulkowitch test showed a faint trace of precipitate. The patient was given 10 mg. of intramuscular ACTH every six hours daily for two weeks. During this time she became alert, less irritable, and her appetite improved. The spleen decreased in size and there was a decrease in total leukocytes. However, after cessation of therapy, she rapidly returned to her previous state. The spleen again increased in size, and repeat roentgenograms of all bones showed no demonstrable change.

A roentgenographic survey of the father, mother, both grandmothers, and the maternal uncle revealed increased density of the bones of the base of the skull in the mother and the maternal uncle.

With the final admission at 2½ years of age, there was marked swelling of the neck and extremities, lethargy, and dyspnea. There was a foul purulent discharge from the nose. Lymph nodes were 5 to 7 cm. in diameter. A loud systolic murmur was heard throughout the precordium. Spleen and liver were enlarged to below the umbilicus. The child expired suddenly the morning following admission after the onset of hemoptysis. An autopsy was performed. Besides generalized osteopetrosis there was hemorrhage into the alveoli of the lungs and in the dura, with focal hemorrhages in the myocardium, liver, and lymph nodes. Extramedullary hematopoiesis was noted in the liver, spleen, lymph nodes, kidneys, and dura.

CASE 2.—E. H., a white female, 1 year 8 months of age, was admitted to the pediatric wards of Gouverneur Hospital on Dec. 26, 1952, because of cough and fever of three days' duration. Birth weight was 2,470 grams, delivery was normal, and prenatal and neonatal histories were uneventful. She was formula-fed and received vitamins and orange juice sporadically. The first teeth erupted at 6 months of age. She had not sat alone, nor stood, by 17 months of age and was noted to have an extremely poor appetite. Because of these she was brought to another clinic where a vitamin preparation was given. Three months later she was brought to our clinic.

Family history: Both parents were born in Puerto Rico and were in good health. Consanguinity in past generations was denied, and there were no other pregnancies. Roentgenograms of the father and mother were negative.

Physical examination revealed an acutely ill, marantic child, dehydrated and tachypneic. Weight was 7,270 grams, head circumference 44.5 cm., chest 40.7 cm., length 71.2 cm. There was ptosis of the left eyelid, reported to have been present since shortly after birth, mild bilateral internal strabismus, and a slight right facial palsy. Remainder of the neurological examination was within normal limits. Eight upper and six lower teeth were present and carious. Examination of the chest revealed signs consistent with bronchopneumonic involvement of the left lung. The liver was palpable 5 cm. below the costal margin. Muscle tone was poor, and there were no gross osseous abnormalities except for increased rigidity of the rib cage. A diagnosis of bronchopneumonia with severe malnutrition and retardation was made on admission.

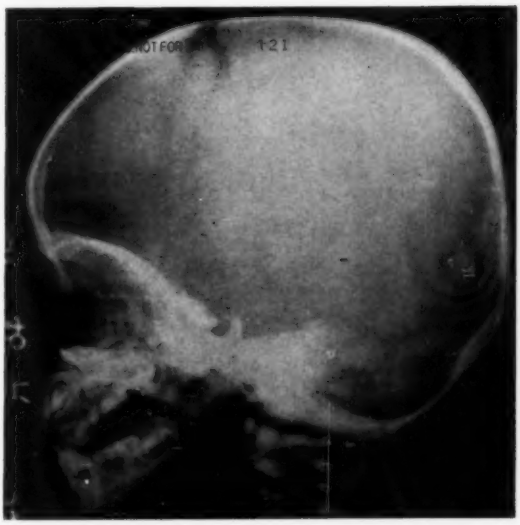

Fig. 4.—Case 2. Patient at 20 months of age. Lateral view of the skull, illustrating the density at the base and the thickening of the clinoid processes.

Initial blood count (after hydration) was: hemoglobin 11 Gm., red blood count 3.7 million per cubic millimeter, white blood count 37,000 per cubic millimeter, with 50 per cent mature neutrophils, 8 per cent metamyelocytes, 4 per cent myelocytes, 28 per cent lymphocytes, 4 per cent monocytes, and 6 per cent eosinophils. The red cells showed considerable anisocytosis and poikilocytosis, and 10 normoblasts per 100 white cells were seen.

Reticulocyte count was 1 per cent. Urinalysis revealed a faint trace of protein and a few white cells per high-powered field. Roentgenograms of the chest and skeleton confirmed the lung pathology but added the unexpected diagnosis of osteopetrosis (Figs. 4 and 5).

Pertinent laboratory data: bleeding time (Duke) 2 minutes, clotting time (capillary) 4 minutes, platelet count 300,000. Stools were negative for ova and parasites, but showed moderate amounts of undigested fat and starch. Sulkowitch test: faint trace. Erythrocyte sedimentation rate (Wintrobe)

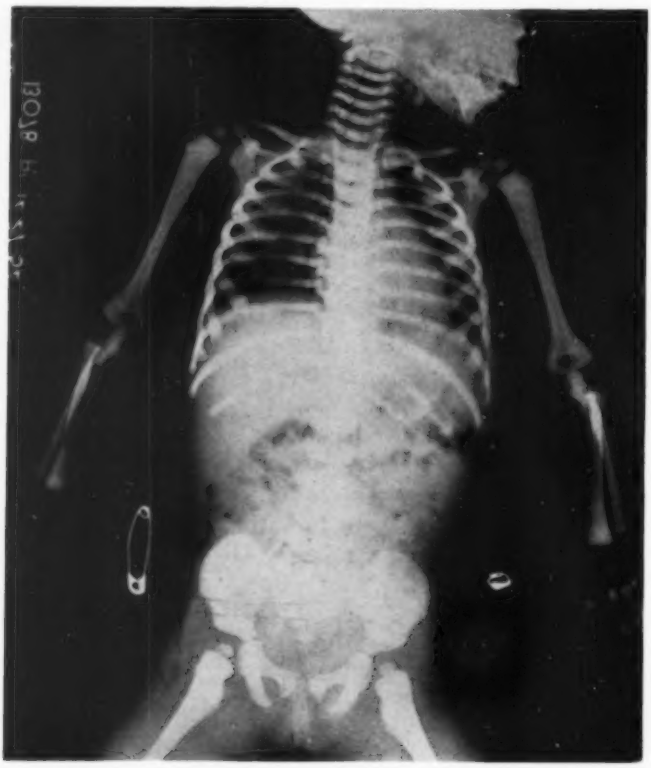

Fig. 5.—Case 2. Note generalized density and clubbing of the proximal humerus and distal radius and ulna.

The infection and dehydration responded satisfactorily to antibiotics and parenteral fluids. During her hospital stay of three months, her physical measurements and weight remained the same, but her appetite improved, and she even began to run about the ward with no evidence of impairment of vision or hearing. Repeated ophthalmoscopic examinations were normal.

11 mm. per hour; Mazzini negative, thymol turbidity, 3 units; cephalin flocculation 2 plus in forty-eight hours; total serum cholesterol, 113 mg.; serum calcium, 10.4 mg.; serum phosphorus 5.2 mg.; serum bilirubin, 0.21 mg.; alkaline phosphatase 2.9 Bodanski units on admission and 8.0 units a month later. Acid phosphatase was 2.9 King-Armstrong units; total protein, 6.9 Gm.; albumin 4.9 and globulin

2.0 Gm. Intradermal tuberculin tests were negative to 0.1 c.c. of 1:1,000 dilution.

She was placed on oral iron medication to see if some degree of hematologic response could be obtained. Reticuloeyte count rose from 1 to 3 per cent in ten days and considerable basophilic stippling and polychromatophilia were seen on smear examination. A month later the hematocrit rose from 36 per cent obtained on admission to 40 per cent. The hemoglobin rose to 12 Gm. and the red blood count to 4.7 million per cubic millimeter. Subsequently, the red cells were normal in appearance, but myelocytes and metamyelocytes were observed in the peripheral blood throughout the hospital stay.

DISCUSSION

Case 1 was of the malignant type, manifesting severe and progressive anemia, thrombocytopenia, hepatosplenomegaly, blindness, and profound bone changes. Early in the course of the disease, because of the hematologic picture, the diagnosis of acute leukemia was made. The presence of periosteal reaction in the long bones helped corroborate this view. The subsequent course and more typical bone changes later confirmed the diagnosis of osteopetrosis.

Case 2 was of the benign type, manifesting, other than malnutrition and retardation, a facial palsy, and a similar, but not as severe, hematologic picture.

Consanguinity, as investigated by Nussey[4] in 1938 was not present in either case. Hereditary factors, however, may have played a role in Case 1 because of the roentgenologic findings in the mother and maternal uncle and the history of miscarriages in the mother and maternal grandmother. These factors were investigated in family surveys by Harnapp,[1] McPeak,[2] and Winter,[5] and are of etiological importance.

Both cases exhibited anemia of varying degrees with persistent shift of the neutrophils to the left. Normoblasts were typically found. Thrombocytopenia, usually terminal, was reported in the cases of McCune and Bradley[6] and Cohen.[7] Significantly, in Case 1, persistent thrombocytopenia and elevated bleeding time were found throughout the course of the disease. Frank hemorrhage occurred at death. The mild anemia of the patient in Case 2 showed satisfactory response to iron therapy. Normoblasts, polychromatophilia, and atypical red cells were not found subsequent to therapy. Similar responses were observed by Kneal and Sante[8] and Vidgoff and Bracher.[9] We feel that this result does not imply specific therapy for this disease; patients with osteopetrosis may suffer from an accompanying nutritional anemia.

Serum calcium, phosphorus and alkaline phosphatase values were not significantly or persistently abnormal in either case. This is consistent with the majority of past reports, unless superimposed rickets is found.[10]

Acid phosphatase may be elevated in this disease. Ensign[11] reported such a finding in the benign form of the disease in an elderly male. Prostatic carcinoma was ruled out by biopsy. In Case 1, a female with the malignant form of osteopetrosis, the serum acid phosphatase was consistently elevated. Since this determination has been rarely performed[12] in previously reported cases, its significance is not known. However, Ensign's values in King-Armstrong units ranged from

10.0 to 12.3, and were well within the range pathognomonic for metastatic prostatic disease. Likewise, our values of 14.3 and 19.2 were significantly high. At present, an explanation is not apparent. Significant acid phosphatase activity has been attributed only to prostatic tissue.[13] This enzyme, however, has been shown to exist in small quantities in liver, spleen, bone, and kidney. Hemolyzed red blood cells may show considerable acid phosphatase activity and are the primary source of error in the laboratory determination. Gutman and Gutman,[14] in 1941, investigated this problem and found that hemolysis and prolonged storage of whole blood samples prior to analysis may falsely raise the results to levels as high as those obtained with metastatic prostatic malignancies. The rejection of hemolyzed samples, as is done in our laboratory, along with repeatedly significant levels tend to eliminate this source of error.

Treatment is symptomatic in all cases, and ACTH was used in Case 1 to determine the effect, if any, of the hormone on the course of the disease. The few subjective and objective changes that occurred while under treatment rapidly returned to the pretreatment state at cessation of therapy. No conclusions can be drawn from this isolated case. To our knowledge, this is the only case of osteopetrosis in which ACTH has been used.

SUMMARY

1. Two cases of osteopetrosis are presented to illustrate the wide range of clinical manifestations. One case is of the benign type, noted as the cause of malnutrition and growth retardation. The other case, with fatal termination, exhibited most of the clinical manifestations of the malignant form of the disease. It is significant that this case was at first misdiagnosed as acute leukemia.

2. Acid phosphatase determinations were elevated in one of the patients presented and warrants future investigation in relation to osteopetrosis.

We wish to thank Dr. Adolph G. DeSanctis for permission to publish the findings of his case.

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Medical Progress

PHYSIOLOGIC STUDIES OF THE BRAIN IN HEALTH AND DISEASE

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IN November, 1950, a symposium on the "Biologic Aspects of Mental Health and Disease" was held at the New York Academy of Medicine under the auspices of the Milbank Memorial Fund. The purpose was to bring together specialists from many disciplines who are working on the common problem of the relation between biology and mental health.

The proceedings of the symposium have been assembled and are now available in book form.[1] The volume of some 650 pages contains, in brief but reliable form, summaries of the more recent studies on the intimate anatomy and physiology of the brain.

The dominant attitude of the participants is expressed by Hoagland[2]: "The statement that all human behavior is a function of the activity of the nervous system and of other body processes is a hardly debatable premise. This is as true of the behavior of normal man as it is of the mentally ill. The so-called functional disorders of psychiatry, of which schizophrenia is an example, presumably are those involving disturbances of brain function, since behavior of a disembodied psyche is a meaningless concept. Such disturbances evidently occur at a molecular level, too subtle to yield directly to present-day analysis by neuroanatomists and neurophysiologists."

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CHEMICAL CONSTITUENTS OF THE BRAIN[3]

Brain tissue is relatively poor in glycogen. That which is present is used for metabolic purposes. The principal constituents of the brain are lipids. They make up from 65 to 75 per cent of the solids in the white matter and 40 per cent in the gray matter. The lipids consist of cholesterol, which exists mostly if not exclusively in the free form and phosphatids, which are compounds containing phosphorus and fatty acids. Another group are the cerebrosides which contain galactose, sphingosine, and a fatty acid. Lipids containing sulfur and gangliosides are also present.

The brain contains nucleoproteins of various types. They are present in combination with lipids as liponucleoproteins. One of these, known as neurokeratin, is trypsin-resistant and pepsin-resistant. A large part of the brain protein is insoluble in water and saline solutions but, in contrast to neurokeratin, is digestible by proteolytic enzymes. Folch and Lees have described a group of lipoproteins to which they have given the name proteolipids because these substances are soluble in chloroform-methanol mixtures and insoluble in water and salt solutions in contrast with other known lipoproteins which are soluble in water and salt solutions but are insoluble in organic solvents.

Folch concludes that the real living tissue of the brain is made up of lipo-

proteins. These are nearer to living protoplasm than either the free proteins or the free lipids.

CEREBRAL CIRCULATION AND METABOLISM

Kety[4] described his studies on cerebral circulation and metabolism of the brain. His experimental technique consists of having subjects breathe a nonmetabolizable gas, nitrous oxide. He measures the difference between the arterial and venous (internal jugular) contents of the gas while equilibrium is being established. In this way he is able to calculate the blood flow to the brain. Once blood flow is known, the oxygen or glucose consumption of the brain can be calculated by obtaining the arteriovenous differences of these substances.

Kety and his co-workers have found that lowering the CO_2 tension in the blood by hyperventilation causes a marked constriction of the cerebral vessels and a reduction in the blood flow; conversely, elevating the CO_2 tension by inhalations of CO_2 produces marked cerebral dilatation and acceleration of cerebral blood flow. The inhalation of 100 per cent oxygen causes mild constriction of the cerebral vessels and slowing of the blood flow; reduction of the inhaled oxygen to 10 per cent produces a large increase in blood flow. Decrease in blood pH also increases blood flow.

A number of drugs influence cerebral blood flow and metabolism. For example, aminophylline (as well as other xanthine drugs including caffeine) causes a constriction of the cerebral vessels and a reduction in blood flow without any change in oxygen consumption by the brain.

In brain tumors, the cerebral blood flow is not reduced in the early stages, presumably because the blood pressure rises. Later on, however, as the spinal fluid pressure rises above 400 mm. of water, a profound and consistent decrease in cerebral blood flow takes place.

The effects of diabetic acidosis and coma, insulin hypoglycemia, surgical anesthesia, and hyperthyroidism on the cerebral blood flow were also studied.

No difference was found between the schizophrenic brain and the normal brain with respect to blood flow, vascular resistance, or oxygen consumption. In this relation Kety states: "Of course one can use this as comfort to that school of psychiatry which believes that schizophrenia is not a disease of the brain but a disease of the mind. On the other hand, one can simply say that this proves that it takes just as much oxygen to think an irrational thought as it does to think a rational one."

BLOOD-BRAIN BARRIER[5]

The concept of a blood-brain barrier arose from the observation of Paul Ehrlich in 1885 that subcutaneously injected aniline dyes, especially the acidic ones, though readily imparting their color to most tissues, failed to stain the central nervous system. The blood-brain barrier manifests itself by a decreased rate of exchange between the two. It is not limited to the choroid plexus but is associated with the entire cerebral vasculature.

Tschirgi points out that all ions, cations and anions, show a markedly decreased rate of penetration from plasma to cerebral intercellular fluid in comparison with other tissues. It is for this reason that the central nervous system volume is so readily

influenced by the intravenous injection of hypotonic and hypertonic salt solutions, a reaction not observed in other tissues. These observations have been corroborated and extended by using radioactive tracer techniques. Thus, following the intravenous injection of tracer doses, bromide, iodide, thiocyanate, phosphate, and chloride equilibrate throughout the body's extracellular fluid within a few minutes except in the central nervous system. Here equilibrium is not reached even in three hours. Sodium and potassium behave similarly, although the rate of penetration is somewhat more rapid for potassium than for sodium.

The observations of Wallace and Brodie indicate that the compositions of cerebral intercellular fluid and cerebrospinal fluid are similar.

Glucose penetrates the cerebral intercellular fluid slowly, protein in only negligible amounts. Labeled water and gases (other than O_2 and CO_2 for which no data exist) enter the central nervous system as rapidly as any other organ.

BRAIN TISSUE RESPIRATION

According to Elliott[6] glucose consumption accounts for from 80 to 90 per cent of the respiration of the brain in situ. During insulin hypoglycemia other substances are burned and this may be the basis for insulin shock treatment. Under these circumstances the material burned is at least partly derived from structural materials and in this way the functional state of the tissue may be altered. Fructose, mannose, and, to a lesser extent, galactose, as well as pyruvate and lactate can be used by the brain in vitro.

The most important noncarbohydrate substance oxidizable by the brain appears to be glutamic acid. It is the only amino acid which the brain can burn.

NITROGEN METABOLISM[7]

The free amino acid fraction comprises somewhat less than 0.5 per cent of the brain weight. Glutamic acid makes up about 35 per cent of the α-amino nitrogen of the nonprotein fraction, a higher concentration than in any other tissue analyzed thus far. Glutamine makes up another 15 per cent. The high concentration of glutamic acid and glutamine is characteristic of the unmyelinated as well as of the mature myelinated brain.

In order to study the origin of these two amino acids in the brain, the compounds were injected intravenously into mice and rats and the organs analyzed at different times after administration. In none of the experiments with glutamic acid did the concentration of this substance in the brain rise. Following glutamine injection, however, the concentration in the brain increased significantly in a number of experiments, indicating that glutamine enters the brain more readily under conditions of elevated blood concentration than does glutamic acid.

Glutamic acid appears to be oxidized by the brain at a more rapid rate than any other amino acid. In brain, as in other tissues, it is a primary member of transamination reactions. Glutamine seems to act as a carrier of glutamic acid and ammonia through the blood-brain barrier and as a store of the two components which may be readily liberated from or recombined to the amide by enzymatic action.

LIPID METABOLISM OF THE BRAIN[8]

Lipids comprise over half of the nonaqueous constituents of nervous tissue.

It is generally considered that their function is structural rather than metabolic since the respiratory quotient in the living animal and in brain slices is one. Recent studies, however, using the isotope-labeling technique, indicate that brain slices are able to oxidize fatty acids. The metabolism of fatty acids is much faster during early development than it is in later life.

Lipids present in the brain are synthesized there and are not brought in from other sources. The lipids of nervous tissue are structurally unique and similar lipids have not been demonstrated elsewhere except under abnormal circumstances.

ELECTROLYTES[9]

The intracellular phase of the brain contains cellular solids, high potassium and magnesium concentrations, and may have little or no sodium and chloride. The extracellular phase has a high content of solids in the form of myelin, sodium, and chloride. A deficit of anions (chloride, sulfate, phosphate, bicarbonate, and so on) is made up to the extent of about 40 meq. by lipids and by phosphoric esters such as adenosine triphosphate.

High external potassium and low calcium increase the excitability of peripheral nerve. The electrical activity of the brain increases with increases in potassium. Magnesium has a depressant action on the central nervous system.

The electrolytes, especially magnesium and potassium, are important in enzyme systems. Potassium is required in high concentrations for functioning of the enzyme choline acetylase. It, as well as magnesium, participates in carbohydrate metabolism.

THE EFFECT OF ANOXIA ON THE STRUCTURE OF THE BRAIN

This subject is discussed by Windle[10] who reviews experiments in his laboratory.

Adult guinea pigs were kept in a decompression chamber of simulated altitude pressure of 23,000 feet (atmospheric pressure, 307 mm. Hg) for six hours each day. Groups of animals were killed after periods ranging from 50 to 500 hours in this rarified atmosphere. No symptoms of brain damage were observed nor were any histologic changes noted.

When the experiments were repeated with pressures further reduced to 225 mm. Hg, simulating an altitude of approximately 30,000 feet, structural changes were found in the brains of the animals which had been in the low oxygen atmosphere for 200 hours or more. The most striking changes were focal areas of degeneration in the cerebellum. Hemorrhages were not observed. Another finding was clumps of leukocytes, resembling emboli, in the blood vessels of the brain and the meninges.

Impairment of memory could be elicted in the animals who had been in the low oxygen atmosphere for 150 hours or longer. Relearning was impossible for the 250-hour group of guinea pigs.

Effects of Asphyxia at Birth.—The fetus and newborn are much more resistant to deficiency in their oxygen supply than is the adult.

Asphyxia was produced in the guinea pig fetus by opening the maternal abdomen at term and occluding the maternal circulation to the placenta. After varying periods of time (4½ to 23 minutes) the fetus was removed and resuscitated.

The young animals without exception showed neurological manifestations—coma, decerebrate states, tremors, convulsive movements, spasticity, paralysis, ataxia, impairment of sensory functions. Structural changes in the brain were observed in every animal asphyxiated for 8 minutes or more. There were multiple capillary hemorrhages with occasional larger hemorrhages and destruction of nerve cells. The destruction was sometimes generalized, but more frequent were focal areas of degeneration in the thalamus, the cerebral cortex, substantia nigra, the tegmentum of the brain stem, and spinal cord. Behavioral changes in animals which had survived and which had the outward appearance of normal guinea pigs were evidenced by the impairment of learning ability.

Windle concludes that animals were produced which survived asphyxiation and showed no residual motor defects and were, in fact, to all outward appearances normal, yet they manifested alterations in mental functioning.

ANOXIA AND BEHAVIOR

McFarland[11] reports on the effects of anoxia on the physiology and biochemistry of the brain and on behavior. He found that anoxia affects visual sensitivity m a r k e d l y. Significant changes occur with atmospheres corresponding to a height of only 5,000 to 7,000 feet.

Since glucose is the chief substance which can be utilized by the brain as fuel, a deficient supply slows the oxidative processes and should have an effect on the brain similar to a reduced supply of oxygen. McFarland found that visual sensitivity showed the same changes after induced hypoglycemia (blood sugar below 60 to 65 mg. per 100 c.c.) as after exposure to a low oxygen atmosphere. Moreover, inhalation of oxygen counteracted, in part, the adverse effect of hypoglycemia. He also found that hyperglycemia reduced the impairment caused by anoxia.

Exposure to carbon monoxide has an effect similar to oxygen deprivation since carbon monoxide combines with hemoglobin, thereby making it unavailable for oxygen carriage. Thus it was found that 5 per cent saturation with carbon monoxide produced a depression in visual sensitivity which was as great as anoxia at 8,000 to 10,000 feet altitude. The impairment caused by 15 per cent saturation with carbon monoxide corresponded to that produced at a simulated altitude of 15,000 to 19,000 feet. Even small amounts of carbon monoxide absorbed from cigarette smoke produced demonstrable effects on visual sensitivity.

McFarland found that mental functions, other than visual sensitivity, were not influenced until anoxia is fairly marked. A significant decrease in the recall of words in standardized tests for immediate memory was demonstrable at 10,000 to 12,000 feet altitude during rapid ascents. Impaired performance on the memory test was not noted in prolonged acclimatization until higher altitudes were reached. Other changes at high altitudes are a general slowing-up of mental functions, a need for greater effort to carry out a task, short attention span, inability in calculations, faulty judgment. There may be feelings of fatigue, euphoria, overconfidence, pugnaciousness, or moroseness. Loss of insight and irrational behavior may be prominent.

The symptoms of chronic mountain sickness resemble in many respects those of patients with chronic fatigue neurosis and aeroneurosis. Similar symptoms are also seen in patterns with cardiac insufficiency, neurocirculatory failure, emphysema, pneumonia, carbon monoxide poisoning, and alcoholism, all of which are associated with cerebral anoxia.

McFarland found that psychoneurotic patients who had marked symptoms of fatigue and exhaustion were more severely impaired in low oxygen atmospheres than were control subjects.

THE EFFECTS OF DRUGS ON METABOLISM AND PHYSIOLOGIC ACTIVITY OF THE BRAIN[12]

Narcotics as a class are, biochemically, inert molecules which undergo little change in the presence of the living cell. It is assumed that their pharmacologic activities are related to their surface activities. Narcotics inhibit enzymic and respiratory processes in the nervous system and, at the same time, reduce oxygen consumption. Since their action may be localized in a small area of the brain, large changes of oxygen consumption might take place in such a small area without being evident when the oxygen consumption by the brain as a whole is studied.

Narcotics inhibit the in vitro oxidation of glucose markedly as well as lactate and pyruvate which are important in carbohydrate metabolism. Morphine (and its derivatives heroin and codeine but not apomorphine) is unique in that it probably acts by competing with acetylcholine for those receptor groups whose combination with acetylcholine is essential for normal functioning of the nerve. During convulsions the rate of cerebral circulation accelerates and oxygen consumption is increased. In convulsions produced by Metrazol the brain content of phosphocreatin is decreased and inorganic phosphate and lactic acid are increased. Oxygen consumption rises leading to a lowering of oxygen tension and possibly increased glycolysis.

Benzedrine or amphetamine, which leads to wakefulness and euphoria, has a high affinity for amine oxidase in the brain. This enzyme oxidizes amines to aldehydes which are highly toxic for the respiratory systems in the brain. Benzedrine appears to halt the activity of this enzyme with a consequent increase in respiration of the brain. Benzedrine doubtless has multiple effects which have not yet been studied.

ENZYMATIC CHANGES IN MENTAL DISEASES[13]

Examples of behavior disturbances associated with interference in the integrity of certain cerebral enzymes are the psychosis of pellagra, in which the brain pyridine nucleotide codehydrogenases are depleted, and the psychotic symptoms that accompany partial inactivation of brain cholinesterase by diisopropyl fluorophosphate. The mental disturbances which accompany the use of sulfanilamide are associated with a reduction in the brain content of carbonic anhydrase.

Pope found the cortical cholinesterase activity of biopsy specimens of the brain, obtained at operation for lobotomy, to be increased in five out of seven patients with deteriorated schizophrenia. According to Meath the

acid phosphatase in the cortical ganglion cells was somewhat reduced in eight out of twelve deeply psychotic patients. Ashby found the brain content of carbonic anhydrase to be low in the majority of patients with schizophrenia, although an appreciable number showed levels approaching those found in normal subjects.

METABOLIC AND PHYSIOLOGIC DISTURBANCES IN THE PSYCHOSIS

Hoagland and his co-workers[2] studied the responses of patients with schizophrenia to stressful situations. Under ordinary circumstances patients with schizophrenia give evidence of adrenal cortical secretion which differs only slightly from that of normal controls. In response to stress, however, more marked differences become apparent. The patient group (consisting of patients with schizophrenia who had been in the institution for an average of two and one-half years) failed strikingly to enhance adrenal cortical output when subjected to stress, as compared with the controls. Such differences from the controls were observed in two-thirds of the schizophrenic patients. Hoagland attributes the lowered responsiveness of the patient group to failure to release adequate amounts and/or types of adrenal hormones in response to stress. Further evidence of the diminished responsiveness of the adrenal cortex is found in the subnormal response of the patient group to injection of ACTH.

Qualitative changes in the responses have also been observed as indicated by differences in the ratios of the various specific manifestations in the patient as compared with the control group.

THE EFFECT OF SHOCK THERAPIES ON THE BRAIN[14]

Insulin Shock.—The brain is particularly sensitive to insulin shock because it is the only organ in the body which obtains its energy almost entirely from combustion of carbohydrate. When carbohydrates are depleted other organs obtain energy by the oxidation of fat. The brain, being unable to do this, must, under conditions of carbohydrate unavailability, slow down its activities. Thus, following the fall in blood sugar after insulin injection, the arteriovenous oxygen difference decreases. Since the rate of blood flow remains relatively constant this represents decreased utilization of oxygen by the brain. At the same time the 10 per second waves in the electroencephalogram disappear, to reappear again when the blood sugar is restored by glucose administration.

The clinical manifestations following a shock dose of insulin show progressive involvement from cortex down to the medulla. The first signs are those of depression of cortical function—disturbances of vision and hearing, drooling, drowsiness and relaxation, and loss of contact with the environment. During the second stage, as the subcorticodiencephalon is involved, there appear motor restlessness, primitive movements such as grimacing, sticking out the tongue, kissing movements, and forced grasping. At this time sensory stimuli usually evoke exaggerated responses. With release of the autonomic system and predominance of the sympathetic come elevation of blood pressure, acceleration of heart rate, flushing of the face, dilatation of the pupils, and inter-

mittent exophthalmos. Convulsions may occur at this stage. The third stage, with release of the midbrain, is characterized by tonic spasms with flexion of the arms and extension of the legs, and torsion spasm. When the medulla becomes involved the patient is pale, the heart rate is slow, the respirations are shallow and slow, the pupils are small, and heat loss is increased.

A similar progression of symptoms is observed following oxygen deprivation. The successive involvement of the different parts of the brain following hypoglycemia or oxygen lack is apparently due to differences in the cerebral metabolic rates of the different parts of the brain. In general, the cerebral metabolic rate (i.e., the demand for oxygen) is highest in the cerebral cortex and lowest in the medulla oblongata.

During hypoglycemic shock the brain falls back on its intrinsic carbohydrate supply of glycogen which, though small, nevertheless acts to extend brain function. A further source of energy is derived from the anaerobic breakdown of adenosine triphosphate and phosphocreatine, both of which are depleted in the brain during hypoglycemia.

Electric Shock and Metrazol Convulsions.—During convulsions respiration ceases owing to tonic spasm of the muscles of respiration and thus no oxygen is supplied to the brain. In addition the energy demands made by the general muscular activity become excessive and oxygen is used up in this way at a rate greatly in excess of normal. As a consequence, in electric shock and Metrazol convulsions as in hypoglycemia, oxidative metabolism is impaired and the anaerobic breakdown of energy deposits in the brain is called upon to maintain function.

The view of Himwich, that cerebral anoxia is responsible for the convulsions in induced hypoglycemia and after electric shock or Metrazol administration, is questioned by Stone.[15] He found that raising the oxygen tension of the cerebral venous blood by administrating oxygen and carbon dioxide did not prevent the changes in brain lactate and phosphates which accompany Metrazol convulsions. Moreover, it has been shown that extreme anoxia suppresses convulsive brain potentials.

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Psychologic Aspects of Pediatrics

PEDIATRIC DISCUSSIONS WITH PARENT GROUPS

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THE pediatric staff of The New York Hospital-Cornell Medical Center considers it desirable for parents of children with chronic illnesses such as nephrosis or diabetes to have as much understanding as possible about the natural history of the disease, the rationale of diagnostic and therapeutic procedures, the relative significance of various symptoms, signs, and laboratory examinations, and the essential problems in management. As one of the methods for achieving this goal, pediatric discussions with parent groups have been held for the past several years. The types of discussions varied and included series of three to five evening meetings each with both parents of children with nephrosis or of children with diabetes mellitus.

In addition, group discussions have been held with either fathers or mothers of newborn infants. The latter consisted of single discussions with numerous groups.

It was found, as anticipated, that such group discussions involved psychologic as well as medical questions. Since the psychiatric training of the pediatricians* who led these discussions was limited, their function was *not* that of group psychotherapists.[1, 2, 3, 4] On the contrary, in the groups as in their individual relationships with parents, the pediatricians served both as medical authorities and as physicians with whom the parents could work through some of their feelings about their sick children, their handling of the children, and their relationship to the medical personnel.

The purpose of this paper is to describe the technique and some of the content of these discussions and to present impressions concerning their value.

GROUP DISCUSSIONS WITH PARENTS OF CHILDREN WITH NEPHROSIS

Two series of evening meetings were conducted in 1952 with parents of children with nephrosis. The mothers and fathers of ten children were invited for each series and most of the meetings were attended by one or both parents of each child. The patients were selected from those who attend the Renal Clinic of The New York Hospital-Cornell Medical Center. The same three staff physicians participated in both series.* In addition, the clinic nurse attended a few of the meetings.

Each series of meetings was introduced by presenting to the parents a

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*All three of the participating physicians were fully trained pediatricians. One (B.K.) had had additional training in growth and development including emotional problems of children; one (L.M.F.) was an experienced practicing pediatrician; one (H.L.B.) had had additional training in the physiology and biochemistry of nephrosis and diabetes mellitus.

*Dr. Henry D. Lauson also participated in the first series of these meetings.

fairly technical explanation of nephrosis in children,[5] including material which had been previously explained to the parents in individual contacts. This portion of the discussion, which was explicitly devoted to health education of the parents, served several other purposes. First, it made it possible for staff and parents to get a comfortable start on the meetings around an objective discussion of topics which were of interest to all present. Second, it offered a framework for subsequent discussion of questions of practical or psychologic significance to individual parents. It was noted that, even during these more technical explanations, there were not only straightforward medical questions but also many that had obvious undertones of anxiety, including a number that concerned themselves directly with fears as to prognosis for life and health in a particular child.

In the course of these meetings it became evident that the attitude of the staff member who initially presented medical explanations affected the emotional atmosphere of some of the subsequent discussions. For example, in the first group of parents the facts known about nephrosis were presented objectively and, partly because there were several physicians in this group of parents, frank statements were made concerning our lack of knowledge about the etiology of nephrosis. This approach set the stage for uninhibited speculations by the parents.

Their serious suggestions as to possible causes for the development of nephrosis in their children ranged from Rh incompatibility to sibling rivalry, and from chilling to improper medical management of respiratory infections in the patient.

It was soon apparent that this atmosphere of "your guess is as good as mine" tended to unnerve the parents. Again and again, in order to reinforce and reaffirm the parents' confidence in the medical profession, on whom they are so dependent, admissions of ignorance by the staff had to be counteracted with definite statements of known facts and established medical judgment.

In order to explore the response to other methods of educating parents about a chronic illness in their child, one group of parents was presented with a booklet[6] which has been prepared for parents of children with nephrosis. The parents' reaction to this reading was fairly homogeneous. They found the booklet interesting and informative but were left with many questions. These were discussed at length at the next meeting of the group. They also felt that such a booklet, although helpful in increasing their understanding of the disease, would have overwhelmed and upset them if they had been presented with it when their child first developed the condition. Because of these criticisms they all felt that physicians should give such a booklet only if they provided ample time for preliminary and subsequent discussion and elucidation. One tangential benefit derived by the parents who read the brochure was that they seemed to obtain considerable satisfaction from seeing some of their own experiences and impressions set down in print.

In the course of several of the meetings the parents described their experiences when their child first developed nephrosis—what was said to them by the physicians whom they consulted, what were their reactions and how the situation was handled. Their reports of these early experiences proved to be fruitful ground for discussion of various points of significance to all the parents.

First, there was evident satisfaction in the very telling of these early experiences, not only to the particular parent who was doing the telling but to the whole group. When painful recollections were being presented by one parent others were ready to offer sympathy and comfort.

One mother broke down and cried in front of the group. The response of the staff and other parents was momentarily limited to reassuring smiles, but the situation was handled by another parent in the group, who relieved the tension by addressing the upset mother by her first name in an easy manner and reminding her that, "All that was a long, long time ago." When one parent told with feeling of a specific problem others would offer reassurance, either by aligning themselves with him and helping him realize that he was not alone in his distress, or by citing their own perhaps worse problem and so providing a perspective in which to look at the situation. "Oh, I remember a time when Tommy ate nothing but peanut butter sandwiches and a little milk for weeks," said one mother to another who was very concerned because she had been unable to keep her child eating an adequate diet for some time.

The parents' relationship to hospitals, doctors and other medical personnel constituted a central theme in these accounts of early experiences. A good deal of overt criticism and hostility toward the doctors with whom the parents had had their initial contacts was expressed. It was apparent that the parents almost held these physicians responsible for subsequent problems of their children. These included problems which they had since learned to be an integral part of the disease process itself, regardless of the way it was managed. However, it also became clear that many of the doctors with whom these parents had had their first contact either did not themselves have sufficient knowledge of nephrosis to feel secure in discussing it with the parents or did not take sufficient time to be as helpful and as reassuring as possible.

One mother in the group told of her distress and anxiety over a series of confused and conflicting statements concerning diagnosis and prognosis with which she was confronted when the child became ill. At the time of the child's discharge from another hospital, the instructions on management of the child at home were given hurriedly and not too clearly. She was later given a detailed diet list according to which bananas were specifically excluded from the child's diet. On the very day on which she received the list, she had given the child three bananas. "I thought I had killed the child," she said.

Such anecdotes almost invariably called forth a host of similar stories from other members of the group. These discussions usually started on a note of individual resentment and diffuse, ill-defined hostility against the medical profession. However, they almost always became general

and finished on a more constructive note of specific criticisms and positive suggestions for improving the situation, such as better training for physicians and more education of the lay public. Some of the parents in the group were in contact with the National Nephrosis Foundation and could there use their drives for constructive action in an organized fashion.

A theme that recurred again and again in the discussions was that of guilt and self-blame by the parents.

One mother had been asking at intervals about any history of falls in the various children represented. Finally another parent turned to her and said, "What is this fall phobia of yours?" At this point the first mother took a deep breath, stood up and said, with much feeling, "I might as well get it off my chest." She then told in vivid detail of an accident in which the child suffered a rather severe fall through the mother's negligence. Shortly thereafter the child developed nephrosis. The mother had apparently always blamed herself for the fall as a possible cause of the daughter's illness. Even the physicians who knew her well had never heard about this. Following this confession, several other parents (without articulating the connection between their stories and those of others) told of events which had occurred before the onset of nephrosis and which they had been suspecting as possible causes for their child's disease. The event recounted ranged from chilling to febrile illnesses managed at home without benefit of medical attention. They all had in common the fact that they involved possible negligence or mismanagement on the part of the parents and had caused feelings of guilt in them.

The accumulation of varied stories and the very telling of them in the group setting seemed to some extent to relieve the parents' guilt feelings and clarify for them the coincidental and noncausal relationship of the particular happening to the subsequent onset of nephrosis. The insight gained through these discussions appeared to be much greater than that achieved by the usual reassurance from the physician. In both series of discussions it was interesting to note that, even as the parents seemed to develop increasing insight into their own reactions to the child and the illness, they persisted in wanting to present their stories and continued to derive obvious satisfaction from these verbalizations.

The history of the fall and its psychologic implications for the one mother cited above had not been previously known to the staff. The staff was also unaware of many other fears as well as a surprising number of misconceptions concerning factual material that was presented in the group sessions. This was true despite the fact that this group of parents had had unusually intensive continuing individual contacts with members of the medical staff over a long time.

Certain practical suggestions came out of the discussions.

For example, it was learned that, with several of the children, the intensive fear of visiting the hospital clinic primarily centered around the dread of venipunctures. These were usually done at the end of the clinic visit and anticipation of them kept the children in a state of mounting tension throughout the morning. It was decided by the group that the venipunctures should be done on arrival at the clinic—a procedure which, on subsequent trial, proved more satisfactory for some of the children.

Throughout these discussions the parents made many helpful suggestions concerning the management of the psychologic problems resulting from the organic illness and the medical experiences of the children.

One mother told with great distress how she had been sufficiently ill-advised to believe that in order to prevent the child from becoming overly upset it was wise not to visit him during sixteen days of hospitalization. The child had become extremely depressed and had given evidence of feeling completely abandoned. He showed increased dependency and fear of being left for some time after the hospitalization.

The description of these reactions made it clear to the group how much a child needs the support of the parents' repeated presence when sick and away from home. Another fact clearly brought out was the great need of these children to play out their hospital experiences as a step in mastering them. The parents reported that the children collected urine specimens from their dolls at home, gave injections and transfusions to their Teddy bears and generally inflicted upon their toys and pets all the indignities to which they had been subjected by the medical personnel.

The children whose parents constituted the second group in this series were quite sick and the prognosis for many of them was poor. This made it difficult for the staff to give objective and satisfactory answers to the recurrent questions about outcome and complications. An attempt was made to be as optimistic and reassuring as possible in the face of the reality of the situation.

It was agreed subsequently by the staff that it is probably not wise to use the group approach with parents of children whose immediate prognosis for health or even life is actually very poor. Since the group approach permits freer expression of fears, and breaks down defenses that are sorely needed by these parents, a group of this composition makes possible situations where anxiety and fear are accentuated and perpetuated. Parents of very sick children can probably be handled more safely by giving support and reassurance on an individual basis.

This viewpoint was reinforced for the staff by the fact that between the second and the third meetings of this group, one of the children unexpectedly developed an exacerbation and died. The parents did not bring this disturbing fact up for discussion at the subsequent meeting, even though some of them were aware of it. The staff had been prepared to have to reassure the group concerning this unfortunate event and its implications for the other children. The fact that in a group the other parents did not feel prompted to raise questions on this subject may have indicated that this situation was too upsetting to be discussed in a group of this type.

GROUP DISCUSSIONS WITH PARENTS OF CHILDREN WITH DIABETES

Two series of meetings with parents of children with diabetes were held. The first was conducted by two staff physicians (H.L.B. and L.L.*) in 1948. It consisted of six meetings held on Saturday mornings. The second series consisted of three evening meetings and was conducted by two staff physicians (H.L.B. and B.K.) with the clinic nurse attending

*Dr. Luigi Luzzatti.[7]

the meetings. The parents of ten children attending the Metabolism Clinic of The New York Hospital were invited to each series. The attendance varied somewhat from meeting to meeting, but at least one parent from each family was present on each occasion.

In keeping with the principles described above, meetings were started with a presentation by the staff of facts and theories concerning diabetes in childhood. A general description of carbohydrate metabolism was given. The disturbances that constitute diabetes, how insulin corrects these disturbances, clinical symptoms and signs of insufficient insulin,[8] and insulin reaction were described. The rationale of urine tests and the interpretation of the results were presented. Because of the many ways in which the families are involved in day-to-day medical management of a child with diabetes more detailed explanations had to be given to these parents. Many of these principles had, of course, been explained to the parents individually during their many medical visits, but it was impressive to the staff members to find that the parents were still in great need of and very receptive to further education concerning their child's condition.

As the discussions progressed, parents repeatedly brought up statements made by doctors, druggists, and in lay publications that had given them false hope or had led to confusion. Most disturbing were reports concerning some person who had been "cured" of diabetes.

This kind of story always constituted a setback to the more constructive discussion of wholesome adjustment to the condition. However, almost always the other parents in the group would refute the stories and theories in a convincing fashion, making it easier for the staff to counter false hopes with more realistic statements of present knowledge.

Although the acute complications that occur in day-to-day care of children with diabetes were discussed by the staff, no effort was made to inform the parents about the late complications in diabetes except as the parents raised specific questions. This protective attitude taken by the staff appeared to be transmitted to some of the parents.

One couple stayed late after one of the sessions to ask a staff member about the increased incidence of tuberculosis in patients with diabetes and explained that they had not wanted to alarm the rest of the parents by their question.

The greater part of one meeting in the second series was devoted to a demonstration and discussion of the technical aspects of giving insulin. A complete set of equipment was brought to the meeting by the clinic nurse. The parents became extremely interested and active in exchanging practical hints and information. They had known of the demonstration in advance and one set of parents brought in the automatic syringe used by their daughter and explained its use to the others.

In these, as in the meetings with parents of children with nephrosis, the staff learned certain facts and psychologic reactions of which they had been unaware, in spite of repeated and extensive individual consultations during which the parents

had been encouraged to express questions, doubts, and anxieties.

In discussing the injections, the necessity for aspirating the syringe was mentioned. Several parents expressed their fear of accidentally giving an intravenous injection. One mother revealed that she had been very apprehensive about her daughter's injections for some time because she had heard of a neighbor who had taken his cat to a veterinarian for a penicillin injection. The injection had accidentally been given intravenously, and the cat's death immediately after the injection had been attributed to the fact that the drug had been given intravenously.

During the discussion of practical management of the child at home, the timing of insulin injections, in relation to time of arising, breakfast, urine collection, and activity, was brought up by some of the parents. Before the staff had the opportunity to suggest the proper time relationships and the range of variability that is safely permissible, the parents described their impressively varied procedures so eloquently that it was evident to all that a very high degree of flexibility was safely possible.

However, here, as in other similar situations, an effort was made by the staff to use the opportunity to describe optimal practice and to give reasons for it.

Throughout both series of meetings a great deal of help was given by the veteran parents in each group to the parents of those children who had developed diabetes more recently. The interaction of the parents in the group appeared to be most active and constructive around those topics that constitute the most challenging problems of caring for children with diabetes. These topics included diet, acceptance of insulin injections, and psychologic reactions of the child to the illness.

The type of diet which should be offered to children with diabetes was the main topic of discussion in many of the meetings. Although diet had been discussed repeatedly during visits with the pediatricians in the clinic, probably no other subject stimulated as much enthusiastic and prolonged discussion in the groups. The New York Hospital staff recommends diets that provide for the normal nutritional needs of the age group without special restrictions because of the diabetes.[8, 9] Concentrated carbohydrates are only interdicted to the extent that large quantities of carbohydrate in place of other nutrients are felt to be undesirable for any growing child. Since it is commonly believed that diabetes is associated with the need to avoid sugar and sweets it is always difficult to persuade individual parents to permit a truly self-selected diet. It was pointed out in the group discussions that, as long as the avoidance of sweets is emphasized in certain families, the children in these families will continue to ask for excessive amounts, whereas if the situation is played down, the children usually select a balanced diet. In the group setting where some of the parents had seen their children's responses first to a strictly limited diet and later to a self-selected one, it was hardly necessary for the staff to offer any arguments or explanations for the use of this dietary regimen. Parents spontaneously presented accounts of their initial distrust of this so-called "free regimen" and of their increasing conviction that the child's general health and progress

seemed to show only beneficial effects. They reported fewer episodes of hypoglycemia and in some cases a decrease in the number of episodes of mild to severe acidosis. To those parents who thought that the children, if left to their own devices, would subsist on a diet of chocolate cookies and ice cream, other wiser members of the group would tell how their children, after an initial period of being abnormally interested in carbohydrate foods, would settle down to a more nearly normal diet.

The psychologic effects of diet restriction were also dealt with adequately in group discussion.

Individual mothers told of their children's psychologic difficulties when on a restricted diet. Others gave vivid and convincing accounts of the adverse psychologic reactions on the part of themselves or friends when kept on reducing diets. One father described the reactions shown by members of his division of the Armed Services when for a period of time they were reduced to a diet consisting exclusively of K rations. They all began to show an absurd preoccupation with food in their thoughts and conversations. Accounts of the practical difficulties in enforcing dietary restrictions were also offered by some members of the parent group.

It was observed during concurrent and subsequent clinic visits by these parents that some of those who had never been able to really follow our suggestions for an unrestricted diet were able to be more relaxed in their management of the children's diets for the first time. However, others, who agreed in principle still were unable to de-emphasize the restriction of carbohydrates in the children's diets.

One mother even articulated this at one of the group meetings. "I know it isn't supposed to do her any harm, but I can't stand to see my daughter put sugar on her cereal." Another mother remarked that in the course of the meetings she had come to believe that carbohydrate restriction was undesirable but that her daughter, the patient, not having attended the meetings herself, could now not be convinced.

The children's reaction to the insulin injections was the theme of numerous discussions in the group. Those parents whose children were undergoing a period of active resistance or overt reaction to the injections were reassured by others with more experience that such phases are inevitable but temporary. Also, reassurance and helpful bits of advice were offered by some parents to those who were concerned because their children were having difficulty giving their own injections. After these discussions, the parents were left with a more positive assurance that the problem of the injections is a nuisance, but one that can be coped with adequately. This appeared to be true even in those who had started with only diffuse anxiousness or an unrealistic hope that an oral preparation of insulin would be available in the near future.

The extent to which the parents were able to accept diabetes in their children varied a great deal. Using the child as the focus, the staff introduced this topic for discussion by inquiring from the parents how they dealt with the child's questions as to why he has diabetes, how long he will have it, and what it means to have it. It appeared clearly that the parents' attitude was the determining factor in the child's ability to accept his condition. Those parents who re-

ported the greatest anxiety in their children were those who themselves voiced the most anxiety, embarrassment, and often guilt feeling about their child's illness.

"Who wants everybody to know their child has diabetes?" asked the mother of one of the more disturbed little girls with diabetes, during the discussion of whether the children tell other children or adults that they have diabetes. Others were able to discuss the matter more calmly and objectively.

Descriptions of the psychologic reactions of the children to their illness brought out quite clearly that the psychologic reactions of a child with diabetes are in no way specific; that aggression and rebellion, withdrawal and shyness, or fairly ready acceptance of the condition are related more to the developmental status and the general adjustment of the child than to the presence of diabetes.

The emotional undertones of the discussions with parents of diabetic children were essentially the same as those with the parents of children with nephrosis. Prognosis for life was not so outstanding a concern of these parents. On the other hand, the hereditary nature of diabetes constituted grounds for a different current of anxiety noticeable throughout the series of discussions.

This was brought into focus early in one of the series of meetings when one pair of parents mentioned that they had two children with diabetes. This fact was obviously upsetting to some of the other parents, who then proceeded to ask many anxious questions around the possibility that their other children might develop the condition. Staff members made every effort to offer reassurance insofar as the facts of the situation permitted.

The fact that none of the other families represented had more than one child with diabetes constituted more effective reassurance. This part of the discussion obviously disturbed some of the parents at least temporarily. However, the staff believed that it was probably less upsetting for parents to learn such disturbing facts in the course of a meeting of this type than under other circumstances where they would not have the opportunity to talk it over with other parents and with their physicians.

Many of the attitudes of the parents of chronically sick children described for the previous group manifested themselves also during the discussions with parents of children with diabetes. Feelings of guilt and self-incrimination for somehow having precipitated the condition in the child (for instance, overfeeding in the early years, or insufficient feeding, or too much sugar in the child's diet before his illness), or for having passed on a hereditary taint (having produced a child with the tendency toward diabetes) were clearly expressed by persons in the group. Again it was the impression of the staff that this kind of anxiety was relieved more effectively by the process of group discussion than by the reassurance an individual physician could have offered.

Ambivalence toward medical and nursing personnel was evident in these parents as well. When one of the parents made a more or less overt attack on the medical profession, it would usually be directed explicitly at doctors and nurses connected with other institutions than The New York

Hospital-Cornell Medical Center. However, in other instances, as in the discussion of the relative advantages of chemical versus clinical control of diabetes, there were discernible criticisms of the local staff and their principles and practices. Usually these expressions of hostility provided the opportunity for some clarification or restatement of principles, and the relationship of the parents to the staff seemed to be strengthened rather than threatened by such exchanges.

GROUP DISCUSSIONS WITH PARENTS OF NEWBORN INFANTS

During the last two years at The New York Hospital-Cornell Medical Center, the obstetrical and pediatric departments have engaged in several joint projects for services to patients and teaching of staff and students. One of the most rewarding of these has been the institution of weekly pediatric discussion groups with the mothers on the lying-in pavilions. Most of these mothers stay in the hospital less than one week and therefore participate in only one such discussion. However, these meetings are part of a continuing program which includes a series of meetings with mothers during the ante-partum period and subsequent individual consultations with the pediatricians whom they consult for well-baby care.

The groups consisted of ten to twenty-five mothers. Discussions were conducted on the ward in an informal fashion by a staff pediatrician (B.K.). The pediatrician who led the discussions always prepared herself by making rounds in the nursery and getting acquainted with current problems so that she could deal with questions in full knowledge of the circumstances that may have prompted them. When questions relating to older children or to individual problems arose, the pediatrician led the discussion back to more general topics concerning the immediate newborn period and the first few months of life. The only exception to this was in the area of sibling rivalry, which was usually brought up for discussion by the multiparous mothers.

In these discussion groups an attempt was made to handle the mothers' suggestions, opinions, and questions in a respectful and permissive manner in the attempt to give them the maximum feeling of self-confidence and trust in their own resources in caring for their child from day to day. At the same time, they were advised to consult their physicians on medical problems.

Each meeting lasted for about an hour. Usually the mothers wished to continue beyond the scheduled time and often they carried on the discussion among themselves after the pediatrician left.

Certain topics of discussion recurred almost regularly. However, the amount of controversy and the intensity of the discussion of any one topic varied from week to week.

In each session there were some questions about the behavior of newborn babies, such as sneezing, hiccoughing, and the startle reaction. These questions were usually answered by the experienced mothers in the group who had observed similar behavior in other infants. The recurrent questions about the many startling physical findings in newborn infants, such as asymmetrical heads,

peeling of the skin, breast engorgement and umbilical hernia were also often answered by members of the group, although at other times the pediatrician was called upon for explanation of these phenomena.

In many of these meetings mothers brought up some of the common misconceptions related to pregnancy, delivery, and subsequent health of the baby.

"If I was nervous and tired while I was carrying the baby, will that mean he will be nervous when he grows up?" asked one worried mother. "I had an awful scare while I carried the baby and I was worried that he might be marked in some way. Can that still happen?" "A friend of mine who was against breast-feeding told me that the mother is more likely to develop milk leg if she breast feeds. What is milk leg?" "Is the fluid that forms in the breast before the milk comes any good for bathing the skin?" "If the baby is born with a caul over his head what does it mean?" "My other baby vomited after he was born and they had to operate. What are the chances of this one having the same trouble?"

These were some of the questions with which individual mothers confronted the group. Almost always other mothers in the group were quick to argue and refute the mistaken notion with information gleaned from their own experience or reading. There was rarely any sense of superiority or condescension apparent in the explanations offered. One got the impression that the group was too close to the problems (all of them being involved with the subjects of pregnancy, delivery, and the care of the newborn babies) to be inclined to laugh off even the most far-fetched concerns on the part of another mother. The pediatrician always attempted to join in the reassurances offered to the mothers asking the questions. In most instances it was the impression of the staff in subsequent contacts with the mothers that many of the fears based on misconceptions had been effectively allayed in the group discussions.

A part of each meeting was devoted to discussion of the practical problems of child care.

Mothers always asked about feeding techniques, skin care, clothing for the baby, fresh air for the baby, traveling with the baby, and many other such problems.

Here again many of the answers and suggestions came from other mothers in the group. Usually there was sufficient multiplicity of opinion and advice offered that the pediatrician could choose and reinforce one or more of the suggestions given by the group. Only rarely, for instance, when one mother made herself spokesman for the group and emphatically advised the others along lines contrary to accepted pediatric principles, was it necessary for the pediatrician to take a firm stand and to give some straight didactic health education to the whole group. Occasional groups would turn the whole session into a question-and-answer period on practical pediatric subjects and rely on the pediatrician to offer most of the answers.

However, at some point in most of the meetings one of the mothers would raise a controversial question and initiate a lively and at times heated discussion.

"Will it spoil the baby if you pick him up when he cries?" "How can you

break the baby from thumb-sucking?" "They say that doctors prescribe pacifiers for babies now. Won't that make them swallow air, and how can you make them give up the pacifier later?"

Almost everyone in the groups had opinions and voiced them on this type of question and the pediatrician could usually act as moderator to the discussion so as to make possible a fairly general acceptance of permissive and flexible attitudes in caring for babies.

At times the session served to release accumulated tension on the ward.

For instance, if one of the babies had been transferred from the nursery to the pediatric ward because of a medical problem, this would be mentioned in some guise during the discussion. One of the mothers might ask a general question, for instance, about the meaning of excessive twitching in a baby: "Is it serious? Could they have prevented it?" Under a thin veneer of academic interest she and her colleagues on the ward might express their apprehensions over the baby's illness or their resentment of the way the problem had been handled by the staff. On these occasions, as in the group meetings with parents of sick children, often the theme of guilt and self-blame would be apparent: "Could it have something to do with me working until a few weeks before the baby was born?"

Staff doctors who had dealt individually with the mother of the sick baby would usually report that these feelings had not been mentioned by the mother in their contacts with her. Floor nurses and nursery personnel were usually sufficiently aware of the emotional atmosphere on the floor to warn the pediatrician beforehand when the mothers were likely to give her "a hard time," indicating that tension and discontentment had been palpably present. Often the discussions appeared to be followed by a general lowering of tension and sense of relief.

DISCUSSION

These group experiences for parents conducted by pediatricians were of such a nature that no precise conclusions can be drawn. Instead an attempt will be made to discuss impressions concerning: (1) The value of this type of group meetings, (2) wherein doctor-patient relationships are different in a group setting than in individual contacts, and (3) the potentialities as well as the hazards of the group approach.

Benefits for the Participating Parents.—There are certain advantages for parents who participate in such discussions. These meetings serve as an occasion for health education. Second, parents, who are selected for having a common problem, derive support from sharing their questions and anxieties with others. Also, the so-called "anticipatory guidance" practiced by pediatricians in their individual contacts with patients can be more effectively carried out in a group by the parents themselves. Thus one parent prepares the others for experiences encountered in relationship to the process of normal growth and development or in the course of the natural history of a specific disease. This kind of preparation seems to be more impressive than predictions and warnings offered by the physician and apparently saves the parent unnecessary anxiety later on.

Another clear-cut effect of dealing with parents in a group consists in adding status to the role of the parent in the parent-doctor relationship. It

is generally the aim of the pediatricians and nurses at The New York Hospital-Cornell Medical Center to conduct their relationship with parents in such a fashion that the parents are able to maintain a maximum of self-respect and dignity as competent adults caring for a well or sick child with the help and guidance of the medical staff. It was observed that the group setting encouraged even the more reserved, less self-confident parents toward self-expression and increased independence. Many of the parents who had remained almost neutral in individual relationships with hospital physicians emerged as distinct personalities only after the group experience.

From the point of view of the parents a number of specific aspects of the group sessions are worthy of consideration. It is understandable that these parents, who undergo years of painful experiences in a state of dependency on members of the medical profession, must have, rightly or wrongly, a good deal of resentment and hostility toward the profession. In most instances, parents cannot express these feelings to physicians and nurses except at moments in which their emotions are stronger than their prudence. It was our impression that the group meetings permitted expression of negative attitudes toward medical personnel in the presence of physicians and nurses and in an acceptable fashion. Where the resentments were based on misunderstandings, explanations could be given by the staff. Where the hostility was based on what appeared to be justifiable grievances these could be voiced in the group discussion with the other parents. This airing of hostile feelings apparently left the atmosphere clear for better and easier doctor-patient relationships.

Another process that took place was of a similar nature. It involved the parents' feelings toward the real enemy—the disease itself (nephrosis or diabetes). By actually participating in extensive discussions of the dreaded condition, the parents appeared to overcome some of their unnecessary, noncomprehending fears and put their reactions to the conditions on a more objective basis. In place of a vague, diffuse anxiety about unknown dangers they acquired an increasingly objective picture of the condition and were able to develop more appropriate reactions to various aspects of the disease. It appeared that an objective and guided discussion of the disease process that had attacked their child gave them a certain degree of mastery over this process in their thinking.

Benefits for the Staff.—For the pediatricians these group experiences were of specific educational value. They provided new insights into child behavior in general, into the reaction of both children and parents to illness, and into the problem of caring for the sick child in the home as well as in the hospital. Finally, the increased understanding and awareness of the physician-patient relationship that developed during the group meetings was probably the single most impressive learning experience for the participating staff members.

It was also of interest for the staff to observe how many specific medical and psychologic facts concerning patients were brought up by the parents during these meetings. Many of these had not been mentioned in

any of the frequent individual contacts with these same parents, even though they had direct bearing on the optimal management of the patient and even though the physicians felt they had offered every opportunity for free discussion. Many points requiring particular emphasis in history-taking were brought out in this manner in the group meetings.

Other topics which had been discussed frequently in individual contacts with the parents and which the physicians thought were accepted and clear to the parents were brought up again in the group meeting. It was instructive for the staff to learn that these subjects appeared to be more effectively communicated in the group than they had been in individual contacts. In this respect, the group meetings seemed to be particularly valuable for discussion of subjects around which the parents had strong feelings and apprehensions such as possible causes for the development of their child's illness, proper diet for the child, and the familial incidence of the disease.

Hazards and Potentialities of Group Discussions.—Although one of the most important functions of these discussions was to reduce the anxiety of parents, it can be inferred from the description of the meetings that the group approach presents some hazards. The outstanding one seemed to be that for some groups (e.g., the second group of parents of children with nephrosis) free discussion may increase rather than decrease apprehension. This undesirable result might occur in any group of parents faced with a reality situation in which the illness of their children is so serious that there could be severe complications or even a fatal outcome. In a group discussion the defenses of such parents are threatened and they are forced to face facts of a painful nature about which, even in the presence of increased knowledge and insight, they are unable to do anything constructive. In these situations, rather than taking some of the pressures off the sick child by relieving parental doubts and anxiety, the group meetings might even have had a deleterious effect on subsequent relationships between parents and children.

In most instances, however, this group approach might be helpful to pediatricians in dealing either with parents of children who present only problems that are part of normal growth and development or with parents of children with a chronic illness. Among the latter might be included such conditions as chronic bronchial asthma, cerebral palsy, mental retardation, chronic anemias, and obesity. In some of these conditions where the patients are of school age or above, the patients, rather than their parents, could be offered the group experience to good advantage. Recently, this kind of meeting was actually suggested by two of the preadolescent girls with diabetes and such a meeting is being planned.

Finally, it is clear that this type of group activity may have important potentialities for teaching pediatricians, nurses, and medical students. The very fact that time is set aside for such meetings should indicate, perhaps more effectively than can be done in any other way, that helping parents of sick and well children is in itself as much a part of pediatric practice as caring for the child.

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Erratum

An error was recently noted in the article by Ethel C. Dunham, M.D., on "Factors Associated With Reduction of Premature Infant Mortality Rates in the United States," which appeared in the JOURNAL, vol. 41, December, 1952. On page 703, eighth line from the bottom in column 2, 14.1 per 1,000 live births should have read *141 per 1,000 live births.*

Comments on Current Literature

PREVENTION OF INFECTIOUS HEPATITIS

DURING World War II, viral hepatitis was recognized by military authorities as a medical problem of the first order. Since that time the disease, particularly the infectious type, has continued to present a serious medical problem in the general civilian population, and considerably more information must be obtained before complete control of the disease process can be achieved. Controlled laboratory investigations have been hampered seriously by the lack of a suitable animal host, other than man. While investigations involving human volunteers have yielded valuable information, such an approach has limitations. Failure of the viral agent to respond to chemotherapeutics and antibiotics has made the management of the clinical disease difficult.

In the search for some practical means for the control of infectious hepatitis, it was logical to consider the efficacy of gamma globulin in possible prevention or modification, since previous experience with antibody-containing blood derivatives, notably gamma globulin, had indicated that passive protection against certain virus diseases could be accomplished by the administration of such immune substances at an appropriate time and in proper dosage. The first report of practical application relative to the use of gamma globulin as a means of prevention in infectious hepatitis was that of Stokes and Neefe[1] in 1945, followed closely by a similar report by Havens and Paul.[2] Since that time a number of publications has appeared describing results which substantiate the original claims.

In a recent issue of the *New England Journal of Medicine* two papers appear which report interesting and convincing data concerning the effective use of gamma globulin in the prevention of infectious hepatitis. The first of these by Alta Ashley[3] presents a careful analysis of epidemic outbreaks which occurred in Maine during the period from Jan. 1, 1952, to April 15, 1953, and in which a total of 671 cases was reported. Two hundred thirty-five households were investigated carefully, and the effect of gamma globulin in preventing secondary cases was subjected to statistical analysis. Gamma globulin, 0.01 c.c. per pound of body weight, was administered to one or more household contacts in 67 of the 235 households studied. Household contacts not receiving gamma globulin were studied as a basis for comparison. Of the 119 secondary cases of hepatitis which were observed in contacts, only three occurred in persons who had received gamma globulin at a time when it could have served as a factor in prevention. These three cases occurred thirty-five, forty-two, and forty-six days following the onset of the last case in the particular household. The over-all attack rate was 16.8 per cent in contacts not inoculated with gamma globulin, as compared with 1.1 per cent in contacts receiving gamma globulin. It was interesting that in these epidemic outbreaks the highest incidence was noted in children of school age, and the source of infection in the schools appeared to be of epidemiologic importance. Likewise, it seemed significant that the largest

number of families having secondary cases was composed of those with three to six susceptible members, and that the highest incidence of secondary cases within the family occurred in families having seven susceptible members. Such observations emphasize the sociologic factors operative in the spread of infectious hepatitis.

The second of the two papers appears in the same issue of the *New England Journal of Medicine*, a study by David Yi-Yung Hsia, Maurice Lonsway, Jr., and Sydney Gellis[4] which offers further information concerning the use of smaller doses of gamma globulin in the prevention of hepatitis in family outbreaks. These authors also report the use of gamma globulin in a dosage of 0.01 c.c. per pound of body weight, their study comprising families whose children were seen from January to July, 1953, in the Outpatient Division of the Children's Medical Center, Harvard University, and at Beth Israel Hospital. By administering gamma globulin to alternate members of 40 families in which a sporadic case of infectious hepatitis had developed, they were able to demonstrate that gamma globulin, even in small dosage, was highly effective in preventing the spread of the disease within the family. In untreated family members, 2 of the 55 exposed adults and 11 of the 40 exposed children developed secondary cases of infectious hepatitis, whereas only 1 case of secondary infection occurred among 37 exposed adults and 57 exposed children who received gamma globulin. In this single instance of secondary infection occuring in the gamma globulin group, the patient showed signs of jaundice six days after receiving gamma globulin.

On the basis of these two epidemiologic studies, the conclusion seems reasonable that gamma globulin is an effective means of preventing secondary cases in family groups having known exposure to infectious hepatitis. The smaller dosage advocated, 0.01 c.c. per pound of body weight, appears to be as effective as that which has been accepted generally, i.e., 0.1 c.c. per pound of body weight, and which had been the prophylactic dose recommended by the Brooks-Hsia-Gellis group on the basis of earlier studies.[5]

Whether the striking results obtained in these analyses are explicable entirely on the basis of passive immunity cannot be stated at present. It is possible that "active-passive" immunity might be operative in permitting a patient to develop antibodies as a result of exposure to the virus at a time when he has sufficient passive immunity to prevent recognizable clinical manifestations. The greater importance of this active immunity in bringing about more permanent protection in a given population is well recognized.

RUSSELL J. BLATTNER

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Correspondence

To the Editor:

In the March issue of THE JOURNAL OF PEDIATRICS, Drs. Shaw and Levin present the concept that whenever the diagnosis of poliomyelitis can be established on clinical grounds muscle weakness can be demonstrated in nearly every case. The editorial comment and the discussion by several workers in the field of poliomyelitis draw attention to the need for uniformity in the criteria for making the diagnosis of nonparalytic poliomyelitis. Apparently the authors have included, as paralytic, cases with "transient muscle weakness," actually a pseudoparalysis due to muscle spasm or pain, as pointed out by Dr. Top. These weaknesses usually clear up after a few days and should not be used as a basis for the diagnosis of paralytic poliomyelitis.

When nuchal resistance and back stiffness are present, the central nervous system has been involved in the disease process. We can assume that there is concomitant damage of anterior horn cells. The function of the motor unit innervated by the damaged anterior horn cell will be impaired either temporarily or permanently, depending upon whether the nerve cell recovers or is destroyed. Therefore, theoretically all patients with involvement of the central nervous system will show some muscle weakness. On this basis we agree with the concept of Drs. Shaw and Levin. Whether this weakness is clinically detectable depends upon how many motor nerve cells are damaged. Clinically we cannot determine the functional loss of a few muscle fibers, especially in a large muscle, so that as a practical clinical problem it seems that the concept of Drs. Shaw and Levin is too broad. For example, in the study at Grasslands Hospital by Moskowitz and Kaplan (J. A. M. A. 152: 505, 1953), referred to by Shaw and Levin, there were thirty-seven cases discharged as nonparalytic poliomyelitis during the 1952 season. This group constituted approximately 30 per cent of the total poliomyelitis admissions. Careful and detailed follow-up examination by the physiatrist one month after discharge failed to reveal any clinical evidence of muscle weakness.

The discussants of the article indicate there is a difference of opinion as to the relative incidence of nonparalytic poliomyelitis. This is due to variations in the interpretation of what constitutes paralysis and the time in the course of the disease at which the final diagnosis of nonparalytic poliomyelitis is made. Both for the protection of the patient and for the uniformity in statistical evaluation, we suggest that the final diagnosis of nonparalytic poliomyelitis be deferred until the patient has resumed his normal physical activity, as emphasized in the article by Moskowitz and Kaplan.

M. BERNARD BRAHDY
EUGENE MOSKOWITZ

To the Editor:

In the report of four cases of boric acid poisoning which appeared in THE JOURNAL OF PEDIATRICS of December, 1953, it was stated that one case resulted from the use of a commercial borated baby powder applied to the infant's excoriated buttocks.

At the time of admission of this baby to hospital, a carefully taken history revealed only the baby powder as a source of boric acid absorption. However, we have recently interviewed the parents of this child again and they now state that, although the borated baby powder was freely applied to the infant's excoriated buttocks, pure boric acid powder was applied to the infected umbilical cord. It is apparent then that, although this represents a proved case of boric acid intoxication, the borated baby powder cannot be implicated per se as the sole cause of intoxication in this infant.

RICHARD B. GOLDBLOOM

News and Notes

On Jan. 7, 1954, Dr. Edwards Albert Park received the "Academy Medal" at the New York Academy of Medicine in recognition of his services as a leader in medical science. The presentation was made by Dr. Alexander T. Martin, President of the Academy.

The following action by the Food and Drug Administration, reported in the *Federal Register* of April 10, 1954, in regard to oil of wintergreen will be of interest to physicians.

Labeling of drug preparations containing significant proportions of wintergreen oil. (a) Because methyl salicylate (wintergreen oil) manifests no toxicity in the minute amounts in which it is used as a flavoring, it is mistakenly regarded by the public as harmless even when taken in substantially larger amounts. Actually, it is quite toxic when taken in quantities of a teaspoonful or more. Wintergreen oil and preparations containing it have caused a number of deaths through accidental misuse by both adults and children. Children are particularly attracted by the odor and are likely to swallow these products when left within reach.

(b) To safeguard against fatalities from this cause, the Department of Health, Education, and Welfare will regard as misbranded under the provisions of the Federal Food, Drug, and Cosmetic Act any drug containing more than 5 per cent methyl salicylate (wintergreen oil), the labeling of which fails to warn that use otherwise than as directed therein may be dangerous and that the article should be kept out of reach of children to prevent accidental poisoning.

(c) This statement of interpretation in no way exempts methyl salicylate (wintergreen oil) or its preparations from complying in all other respects with the requirements of the Federal Food, Drug, and Cosmetic Act.

Book Reviews

Neurosurgery of Infancy and Childhood. Franc D. Ingraham and Donald D. Matson, Springfield, Ill., 1954, Charles C Thomas, 456 pages. Price $15.00.

As stated in the Preface, this volume is not designed as an encyclopedic text of reference nor as a forum for discussion of various methods of treating neurological disorders of children. It describes simply and with unembellished detail the diagnostic and therapeutic methods which have stood the test of the authors' personal experiences in an active neurosurgical service at the Boston Children's Medical Center.

One-third of the book is devoted to congenital anomalies and hydrocephalus. The indications for surgery in infants with meningocele or myelomeningocele, with or without paralysis of the extremities and sphincters, are presented with full awareness of the surgeon's social and ethical responsibilities. The brilliant results which follow early recognition and surgical excision of dermal sinus tracts stand in sharp relief against those cases which are overlooked until infection spreads along the extension of such tracts into the cranial cavity or spinal subarachnoid space. In primary craniostenosis the gloomy end results of compression of the brain can be avoided by early craniectomy, with an excellent prognosis if the condition is recognized and treated during the first year of life. The authors present a concise summary of the factors producing hydrocephalus, the diagnostic procedures which should be performed to determine the type of hydrocephalus, and summarize their experiences with the various surgical methods which have proved of value. Among these are included choroid plexectomy, and shunting procedures such as ventriculo-cisternostomy, arachnoid-ureterostomy, and ventriculo-ureterostomy.

The section on trauma includes two important chapters on the diagnosis and treat-

ment of extradural hematoma and the more commonly encountered (when looked for) subdural hematoma of infancy. That increased interest results in more frequent diagnosis is illustrated graphically by a chart of yearly incidence. The authors' discussion is based on their experience since 1937 with 319 patients under 2 years of age treated for subdural hematoma. Their recommended course of treatment consists of repeated subdural taps, exploratory burr holes to establish presence or absence of a membrane, and craniotomy with excision of the hematoma membranes.

Intracranial tumors in infancy and childhood are presented in detail, this section being based upon experience with 313 cases. The high proportion of gliomas (80 per cent) and the higher incidence of infratentorial lesions (60 per cent) in this series fits in with the general experience of other clinics. Since the majority of tumors in children occur below the tentorium, symptoms and signs of posterior fossa tumors are clearly presented, stressing the early significance of vomiting and intermittent headache of insidious onset. Among 185 posterior fossa tumors, there were 56 astrocytomas which have a poor outlook. In medulloblastomas temporary improvement follows surgery and x-ray therapy. Gliomas of the cerebral hemispheres were found in 42 cases. The commonest nongliomatous tumor was the craniopharyngioma (21 cases). The suprasellar location of this lesion makes it suspect by signs and symptoms referable to increased intracranial pressure, visual disorders, pituitary and secondary endocrine dysfunction, and disturbances of hypothalamic function. In the majority of the authors' patients with craniopharyngioma, there was tell-tale roentgenographic evidence in the form of enlargement or distortion of the sella and suprasellar calcification. Pre- and postoperative supportive treatment with cortisone and ACTH is outlined.

Intraspinal tumors are relatively uncommon in childhood. Among 63 cases of the present series there was a significant preponderance in the cervical and lumbar regions due to the large number of "congenital" tumors in these areas; these include teratomas, teratoids, and dermoid cysts.

The section concerned with infection is brief. Of major importance is the complication of subdural effusion in meningitis. The authors found this complication in 50 cases, half of which followed meningitis due to *H. influenzae*. They recommend that treatment follow the same course as in traumatic subdural hematoma.

In their discussion of congenital vascular malformations, the authors advise that angiography be performed in cases with repeated focal seizures without intracranial pressure, high fever, history of trauma, or other apparent abnormality. Convulsive seizures, particularly when they are consistently focal, are a common symptom of cortical arteriovenous abnormalities. Although an audible bruit may be present in such cases, it is stressed that absence of a bruit does not rule out a vascular anomaly. With regard to surgical treatment of convulsive seizures due to other conditions, it is pointed out that there should be evidence of a well-localized pathological process and operation considered only when the seizures cannot be controlled by thorough, supervised trial of anticonvulsant medication.

The concluding chapter on pediatric neurosurgical anesthesia is eloquently short. To avoid anxiety, struggling, and hypoxia during induction, Avertin is recommended as a basal analgesic. Pentothal, curare, and intraspinal agents have not found favor. In the authors' experience, the safest and most satisfactory anesthetic agent for critically ill children is ethyl ether and oxygen administered by careful endotracheal insufflation.

This is a well-organized and well-illustrated review of the many practical neurosurgical problems which occur in children, and deserves a readily accessible spot on the desk of every pediatrician. The book will also be a welcome source of reference for the neurosurgeon whose pediatric experience may be relatively limited.

HENRY G. SCHWARTZ

Die Mund- und Rachenkrankheiten. Prof. Dr. C. R. Griebel, Stuttgart, 1954, Georg Thieme Verlag, 140 pages. Price $6.90.

A monograph by Prof. Griebel of Bad Nauheim. After a discussion of the anatomy and methods of investigation, the author discusses, in turn, diseases involving

the teeth, mucous membranes, tongue, salivary glands, and the lymphatic tissues. The text is well illustrated and makes a useful reference book for diseases of the mouth and pharynx.

The Pre-adolescent Exceptional Child. Proceedings of the 35th Conference of the Research Clinic of the Woods Schools, Langhorne, Pa., 66 pages.

The proceedings of the Conference held in Philadelphia in April, 1953, contain three papers: "Diagnosis of the Various Syndromes of the Retarded Child" by A. A. Silver of New York; "Treatment of the Mentally Retarded Child" by G. H. Walker of the Polk State School; and "Treatment of the Emotionaliy Disturbed Child" by Leon Eisenberg of the Johns Hopkins Hospital. A panel discussion on "What's Ahead?" in which a number participated concludes the proceedings.

Your Wonderful Teeth. G. Warren Schloat, Jr., New York, 1954, Charles Scribner's Sons. Price $2.25.

A beautifully illustrated book of about 50 pages with the purpose of interesting young children in the importance of their teeth and the care of the teeth. The author, an artist and photographer, has taken a remarkable series of photographs of his own children as they were growing up, which illustrate the book. The advice given through the captions of the photographs and drawings is sound, and the book, because of its pictorial interest, should be of definite educational value for young children.

Manual of Clinical Mycology, Philadelphia, 1954, W. B. Saunders Co., 456 pages.

The manual of mycology has a group of authors interested in this field: Conant, Smith, Baker, and Callaway of the Duke University School of Medicine, and Martin of the Bacteriology Section of the Communicable Disease Center in Georgia. A first edition was published ten years ago. Many new facts have developed in mycology in the last decade as the incidence of coccidioidomycosis and histoplasmosis. New skin-testing antigens have been found to be of value in some types of mycotic infection. In addition to the clinical discussion of the various mycotic diseases, the text contains a chapter on laboratory methods. With the increasing importance of mycotic diseases, the volume is a useful addition to medical literature. It is excellently illustrated.

1954 Current Therapy. Edited by Howard F. Conn, Philadelphia, 1954, W. B. Saunders Co., 898 pages.

The 1954 edition of *Current Therapy*, which is published yearly, continues to present the latest as well as the more thoroughly tested methods of treatment. The present volume contains methods by 192 new contributors. It is a satisfactory and useful reference volume on therapy for the busy practitioner who has difficulty in keeping abreast of the constantly increasing number of new drugs.

Mayo Clinic Diet Manual. Second edition. Philadelphia, 1954, W. B. Saunders Co., 217 pages.

An excellent diet manual by the Committee on Dietetics of the Mayo Clinic. In addition to the usual list of hospital diets for specific medical and surgical conditions, considerable space is given to diets for children. These include diets for infants; general, soft, and liquid hospital diets for children of different ages; a dietary program for celiac disease; the ketogenic diet for epilepsy; restricted sodium and low protein diets for children with chronic renal disease; and diabetic diets for children. The material is well arranged and well presented in a useful, practical form.

Editor's Column

ACCIDENTS IN CHILDREN

WE WERE recently asked why the JOURNAL had been stressing accidents in children, both editorially and by the publication of reports of accidental poisoning. The answer is a simple one. It was in part well stated recently by Jacobziner and Rich,[1] who pointed out not only that "accidents rank *first* as a cause of death from 1 to 24 years of age" but that "in 1952 in New York City . . . more children in the age group 1 to 5 years died from fatal accidents than from diphtheria, whooping cough, measles, German measles, poliomyelitis, rheumatic fever, meningitis, and tuberculosis combined."

As a matter of fact the JOURNAL has not been stressing accidents to children in general, but rather accidental poisoning in young children due to medicaments, as these are accidents for which the medical profession and the pharmaceutical houses have a definite responsibility both as to cause and prevention. Accidental poisoning from drugs, such as aspirin, ferrous sulfate, boric acid, barbiturates, etc., carelessly left available to ingestion in quantity by young children, is an important matter for pediatric discussion. A part of the blame for such accidents must be placed on physicians for failure to stress their potential danger, and part to the failure of pharmaceutical houses to call attention on the label to this danger.

A few months ago in discussing editorially[2] a report of the incidence of accidental poisoning in young children in England and Wales, we estimated that, based on comparative population figures, some 900 deaths had probably occurred in runabout children in the United States during the same period. This figure was based on the assumption that "there is no reason to believe that the incidence is much greater or less per 100,000 of population in the United States." How wrong we were. Stimulated by Swinscow's[3] report, Dr. Katharine Bain of the Children's Bureau made a study of the vital statistics records of the Census Bureau in Washington for the similar age group over a like period. This was presented at the meeting of the American Pediatric Society last month and appears on page 616 of this issue. Dr. Bain comes forth not with 900 as we estimated, but with the shocking figure of 3,659! Why the incidence rate should be over four times higher in the United States than in England and Wales is difficult to explain or understand. Is carelessness so much greater in American homes than in English?

In studying Bain's figures we note a marked increase in the number of fatal cases of poisoning from hydrocarbons, such as kerosene and gasoline, when compared with the British figures. In Swinscow's report these accounted for only 4.5 per cent of the total fatal accidents, while in Bain's series 25.4 per cent were due to kerosene and gasoline. Hydrocarbons were

particularly frequent as a cause of fatal poisoning in the southern states. Drugs were responsible for 62 per cent of the deaths in the British cases and for 33 per cent of the American cases. However, when we take into consideration the higher death rate from accidental poisoning in the United States, the rate from drugs per 100,000 of population is higher.

Jacobziner and Rich[1] recently presented an interesting discussion of accidents in the preschool child and the methods which the Bureau of Child Health in New York City is developing in its preschool age clinics for accident prevention and home safety. As a part of their work an epidemiological study was made of 1,000 nonfatal accidents which occurred in 981 children. More occurred in males than in females. Serious accidents, defined as those requiring medical aid, were most frequent in the 24- to 35-month age group. Seventy-five per cent of the serious accidents occurred indoors and 23.9 per cent outdoors. Sixty-five and four-tenths per cent of the 1,000 accidents occurred in the child's own home and only 4.7 per cent on the playground. Falls accounted for 45.2 per cent of all injuries, and burns for 21.3 per cent. Burns were most frequent in infants under 1 year, and the incidence of falls decreased rapidly at 5 years of age. Forty-four per cent of the accidents occurred between 12 noon and 5 P.M. Such studies give a sound basis for indicating the points that must be stressed in developing an educational campaign for home safety and accident prevention.

A tremendous decrease in the fatality rate of the infectious diseases has taken place in recent years as the result of specific immunization measures, and the advent of the sulfa drugs and the antibiotics. As a result, many of the urgent problems which faced pediatricians only a few years ago have to a large extent disappeared. Thus time and opportunity are now available to take up the problems related to childhood accidents which today are a leading cause of death. While perhaps their frequency may be no greater than a decade or so ago, their relative importance has tremendously increased in mortality statistics. This is the answer to the question that was asked.

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CORTISONE IN THE PREVENTION OF RHEUMATIC CARDITIS

AGAIN the JOURNAL presents in this issue a discussion of a controversial subject. Some cardiologists feel that ACTH and cortisone have a definite influence in the prevention of permanent cardiac damage in rheumatic fever, while others consider this as unproved, and that such a conclusion is merely a post hoc argument. They point out, as is common experience, that some cases of acute rheumatic fever with carditis treated with salicylates clear up without permanent cardiac damage. Heffer and associates, in a discussion in this issue, report on the use of cortisone in amounts to produce physiological effects. They feel that it has an influence in preventing permanent cardiac damage in patients developing carditis

in their first attack of rheumatic fever. Whether their conclusion is right or wrong requires the study and observation of series of similarly treated patients by other workers.

Reports such as this always lead to lengthy discussions by the Editorial Board. Some readers, the Board realizes, unfortunately take the viewpoint that the acceptance and publication of a report or study of a suggested therapeutic measure or procedure means approval and endorsement by the JOURNAL and its Editorial Board. This is not true. In this particular instance the thought was expressed by several that it might lead to the indiscriminate, unwise, and uncritical use of cortisone in patients with rheumatic fever, for example, by the practitioner in the home. We do not believe that at present it should be used except in hospitals, where the patients can be closely observed, studied, and followed, with the purpose of throwing further light on its value or lack of value in preventing permanent cardiac damage. It is a series of well-studied and well-presented cases, and the Board feels that every possibility of prevention should be investigated and explored when it comes to such a crippling disease as chronic carditis in a rheumatic child. The JOURNAL has an obligation to present new ideas or therapeutic suggestions which have the possibility of advancing medical practice, even if at times they are contrary to the thinking of the members of the Board.

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